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Original Communications

NONSPECIFIC PNEUMONITIS OF THE LEFT UPPER LOBE (SIMULATING THE "MIDDLE LOBE SYNDROME" AND PRODUCING AN EARLY SUPERIOR PULMONARY SULCUS SYNDROME)

REPORT OF CASE*

WILLIAM M. ASHE, M.D.,† JOHN R. MCDONALD, M.D.,‡ AND
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ROCHESTER, MINN.

THIS report deals with a case in which nonspecific pneumonitis was situated in the apex of the left upper lobe. The lesion produced some symptoms characteristic of the superior pulmonary sulcus syndrome, and the pathologic findings in the resected left upper lobe resembled those of the so-called middle lobe syndrome reported by Graham, Burford, and Mayer[1] as a disease entity.

REPORT OF CASE

On April 12, 1949, a 33-year-old married woman who was six months' pregnant, registered at the Mayo Clinic because a lesion in the apex of the left upper lobe had been discovered by her family physician. Review of her past history revealed no previous serious illness. Each spring for the past seven or eight years she had had mild pyrexia which had been controlled easily by administration of pyribenzamine. In addition, she stated that during the previous winter she had had "bronchitis" on two occasions. On each occasion, the "bronchitis" had lasted about ten days and had been characterized by fever and a tight paroxysmal type of cough that became productive as she improved. In the course of the "bronchitis" her temperature had been as high as 103° F. She had been treated with sulfonamide drugs during each episode, and complete remission of her symptoms had occurred.

Six weeks before the patient came to the clinic, a nonproductive persistent cough had developed and had been accompanied by pain over the anterior portion of the left side of the thorax. During the next four weeks these symptoms had become progressively worse and the pain had begun to extend to the left shoulder and down the medial aspect of the left arm to the elbow. The cough had become productive of about 1 tablespoonful of clear phlegm in twenty-four hours, and on one day her temperature had been 101° F.

About a week before the patient came to the clinic, her family physician had found a "lump" in the left supraclavicular region. Pressure on this "lump" had caused the pain to extend to the left shoulder and down the left arm. During this time, she had received 1,200,000 units of penicillin without any noticeable benefit. Roentgenographic ex-

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*Abridgment of a portion of thesis submitted by Dr. William M. Ashe to the Faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Surgery.

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probably indicative of superior pulmonary sulcus tumor (bronchiogenic carcinoma) of the left upper lobe. It was felt, however, that the presence of pulmonary tuberculosis should be ruled out.

Bronchoscopic examination did not disclose any abnormality, and cytologic examination of the secretions and smears obtained from the left upper lobe bronchus did not reveal any malignant cells.

After bronchoscopy, the patient had irregular uterine contractions, which she characterized as being typical of labor pains. These were controlled by rest in bed and by the administration of sedatives, stilbestrol, and corpus luteum extract.

In spite of the fact that bronchoscopy and examination of the sputum did not reveal that the lesion in the left upper lobe was of a malignant nature, it was felt that the presence of the mass in the left supraclavicular area, the abnormal density in the roentgenogram of the left upper lobe, and the symptoms necessitated an exploratory thoracotomy.

The patient was kept in the hospital for a few days for preoperative preparation because of the anemia and threatened miscarriage. She received three blood transfusions of 250 c.c. of whole blood each time, and therapy was continued to prevent premature labor.

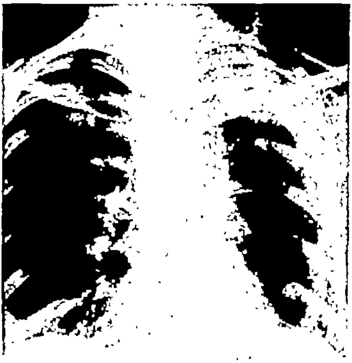


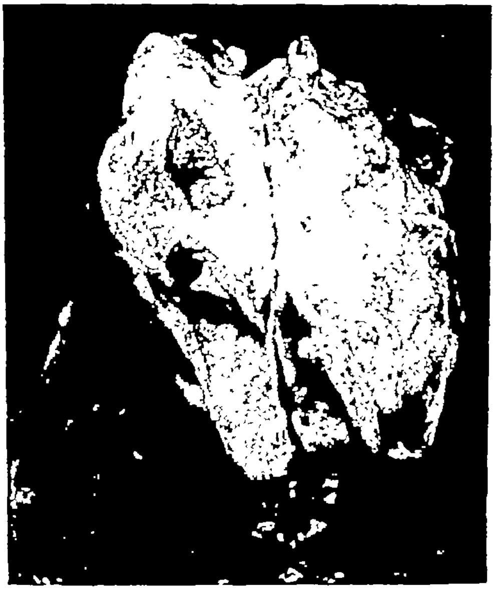
Fig. 2.—Postero-anterior roentgenogram of thorax made on the thirteenth day after lobectomy. The left lower lobe is expanding to fill the defect caused by removal of the upper lobe.

On April 19, 1949, an exploratory thoracotomy was performed by one of us (Clagett) by means of a left posterolateral incision and resection of a long posterior segment of the fifth rib. A firm, nodular lesion was found in the extreme apex of the upper lobe of the left lung. The mass was firmly adherent to the thoracic wall. It was grossly suggestive of a superior sulcus tumor of a malignant nature. The lung was dissected free through an extrapleural line of cleavage without difficulty. The upper lobe of the left lung was removed by dividing and ligating the hilar vessels individually. The bronchus was closed with a single row of interrupted silk sutures, and the bronchial stump was covered with pleura. The thoracic wall was closed in layers, and two intercostal catheters were inserted for temporary closed drainage after instillation of 200,000 units of penicillin into the pleural space.

The convalescence was uneventful except for occasional irregular uterine contractions in the immediate postoperative period. The patient was permitted to return to her home on the fourteenth day after the operation. On the thirteenth day after the operation, roentgenographic examination revealed that the lower lobe of the left lung was expanding to fill the defect caused by excision of the upper lobe (Fig. 2).

In the extreme apex of the excised lobe there was a very firm nodular lesion which measured 6.5 by 4 by 4 cm. (Fig. 3). The pleura over the lesion was firmly adherent, greatly thickened, and reddened. The remainder of the lobe was aerated and grossly normal.

On section, the involved portion of the lobe was firm and whitish yellow in appearance. The involved firm and contracted segment corresponded to the posterior apical division of the left upper lobe bronchus. The lobar lymph nodes were enlarged and moderately firm. They closely encircled the left upper lobe bronchus and its divisions.



3 —Firm, whitish-yellow patch of pneumonitis in the apex of the left upper lobe. This represents an advanced stage of pneumonitis.

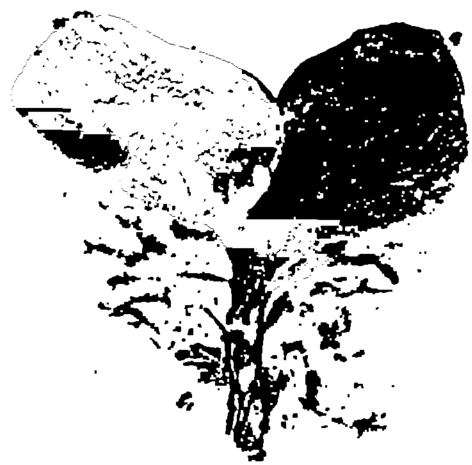


Fig. 4.—Mediastinal surface of left upper lobe. The hilar lymph nodes are enlarged, and normal appearing bronchi end as a "pencil point" in the region of pneumonitis.

The bronchi showed no evidence of extrinsic bronchial compression, endobronchial lesion or bronchiectatic changes. Rather, the involved posterior apical bronchus and its branches were gradually reduced in caliber to a point, similar to a ''pencil point,'' which ended in the region of the lesion (Fig. 4).

Microscopic examination of the consolidated portion of the lobe revealed inflammatory cells including polymorphonuclear leucocytes, lymphocytes, eosinophils and lipoid-filled phagocytes, and abscesses. In this portion, there was marked fibrosis (Fig. 5). In the adjacent pulmonary tissue the alveolar spaces were dilated and congested with serum, erythrocytes, and large mononuclear phagocytic cells.

The bronchi were not filled with secretion. In this respect, the microscopic appearance differed from that resulting from obstructive pneumonitis[2] in which the bronchi often contain secretion and cellular debris.

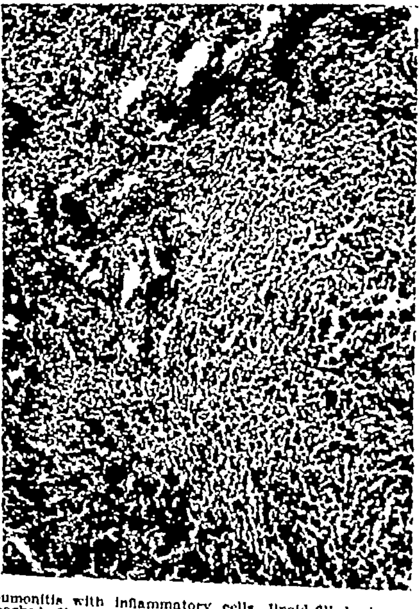


Fig. 5.—Diffuse pneumonitis with inflammatory cells, lipoid-filled phagocytes, abscesses, and marked fibrosis (hematoxylin and eosin stain, ×100).

COMMENT

Pancoast's conclusions[3, 4] concerning the syndrome bearing his name are well known. This syndrome has since been known by various
and it has been established by numerous authors that a
region of the thoracic inlet will likely produce the cha
plex of involvement of the brachial plexus and the ce,
and that bone will be involved as the mass enlarges
and Watson,[5] in 1946, in a comprehensive review of the
syndrome, found 134 reported cases plus an additiona
discussions at medical meetings. They also reported 1
observed. All features of the syndrome were not pr
case, but the authors said that the disease had been

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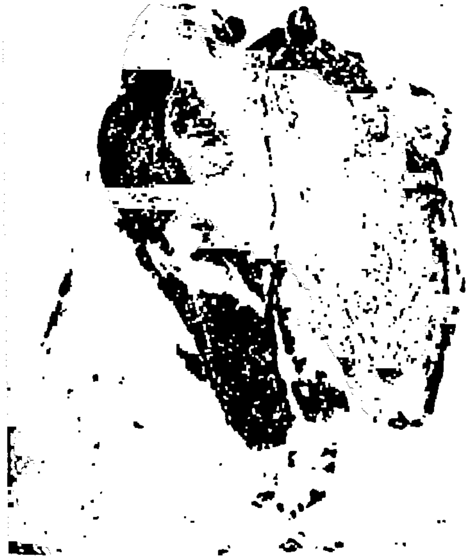

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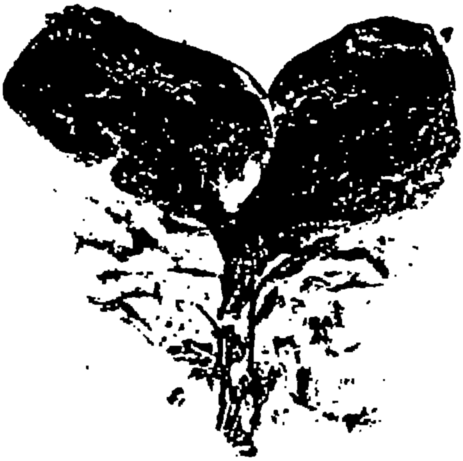

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EXFOLIATIVE CYTOLOGY

An Adjunct in the Diagnosis of Bronchiogenic Carcinoma

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BRONCHIOGENIC carcinoma has elicited considerable attention since this form of carcinoma has shown a steady increase in incidence during the past thirty years, while the incidence of carcinoma of the skin, uterus, stomach and duodenum, or liver and gall bladder has remained approximately the same.[1, 2] Thus it was reported by Jeuther and associates[3] that in a series of 35,819 autopsy reports collected in various hospitals in Prague, bronchiogenic carcinoma constituted only 1.3 per cent of all the carcinomas encountered at post-mortem examination in 1894, and rose to 12.7 per cent in 1943. Although various theories as to the reason for such an increase have been advanced, no conclusive evidence has been produced to incriminate any one environmental factor other than exposure to radioactive ores.[4] As long as the direct agent remains obscure, only early diagnosis and early treatment can serve to check the mortality from this disease.

Much has been written about the nonspecificity of symptoms, the nonrevealing findings on physical examination, and the deceptive radiologic findings which frequently accompany the early lesions, particularly the early peripheral lesions. As a result, many bronchiogenic carcinomas remain undiagnosed until too far advanced for surgical intervention. Overholt[5] pointed out that in a series of 165 patients with lung cancer, 63 per cent were misdiagnosed at initial examination, and were treated for long periods of time for tuberculosis, unresolved pneumonia, lung abscess, bronchitis, etc. An average of nine months elapsed from the time the first symptoms were noted until the diagnosis of carcinoma was definitely established, the physician having been responsible for five months of the delay. With the average survival from the first symptoms in untreated cases only 10 to 14.5 months,[6] it is obvious that earlier recognition is imperative. Bronchoscopy has certainly contributed a great deal in this respect since it has made possible tissue diagnosis in 60 to 70 per cent of the cases.[4, 7] Churchill admitted, however, that these figures are based on series heavily laden with advanced and inoperable lesions. Björk,[8] who has broken down his statistics into more detailed categories, revealed that in centrally located tumors the bronchoscopic biopsy was positive in 86 per cent of

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the cases, in intermedially located tumors only 30 per cent, while in peripherally located tumors no positive biopsies were available. These statistics indicate that further diagnostic procedures, which would more consistently reveal tumor regardless of its location in the tracheobronchial tree, would serve as valuable adjuncts in the diagnosis of this disease.

The microscopic examination of sputa and bronchial aspirations has become recognized as a constructive adjunct to diagnosis. It has been demonstrated that carcinoma cells can be identified in smears of expectorated or aspirated bronchial secretions by virtue of the exfoliation of these cells into the lumen of the bronchi (Figs. 1, 2, 3, and 4). The classic report on this work is that of Wandall,[8] who was able to recognize malignant cells in the sputa in 84 per cent of his cases of bronchiogenic carcinoma. In our experience with the cytologic diagnosis of lung lesions, we were able to make some further observations pertaining to the use and abuse of this method. We have thoroughly reviewed the literature and studied smears from a series of 270 patients who were admitted to Chest Service and General Medicine Service of Barnes Hospital for diagnosis and treatment of lung disease. Included in our group are 100 cases of bronchiogenic carcinoma, 5 metastatic lung tumors, 2 recurrent bronchiogenic carcinomas, 4 adenocarcinomas, grade 1 (so-called bronchial adenoma), and 159 benign lung lesions.

MATERIALS AND METHODS

We began our study by organizing the work so that we could demonstrate the practicability of routine cytologic examination on patients admitted to the hospital for diagnosis of lung disease. We requested that fresh, early morning sputum specimens—not night cups or 24-hour collections such as are used for acid-fast studies—be sent to our laboratory on three consecutive mornings plus any available bronchial secretions aspirated at bronchoscopy. Whenever cells suggestive of but not absolutely consistent with the criteria of malignancy were found, a special request for additional material was made. Naturally it was not always possible to obtain the desired number of specimens. Four slides were prepared from each specimen. These were fixed in a solution of equal parts of ether and 95 per cent alcohol as soon as the material had been smeared and the film on the slide was still wet. After one hour of fixation these were stained according to the modified Papanicolaou procedure described by Gates and Warren.[9] This stain was preferred to the regular hematoxylin and eosin since the relative transparency of the cytoplasm allows greater clarity of detail and the polychrome tinctorial qualities are more restful on the eyes during the long periods of time required for interpretation of the slides. The slides were then screened, a procedure which usually took 15 minutes per slide for the negative cases, often much longer for borderline cases. Only three interpretive categories were used: positive, negative, and suspicious; an intense effort was made to eliminate the latter category whenever possible. Many of the suspicious and all of the positive cases were reviewed by Dr. Ackerman before a report was made to the clinician.

Fig. 1.

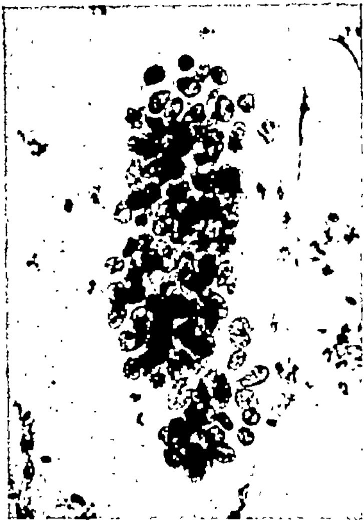
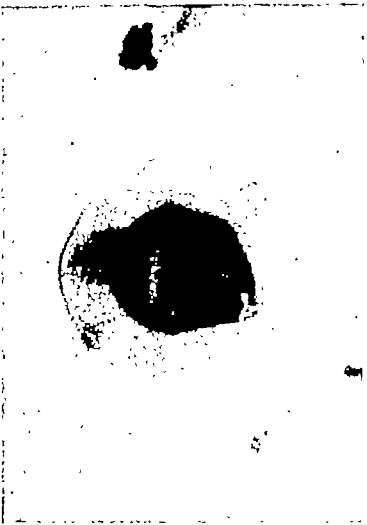


Fig. 2.



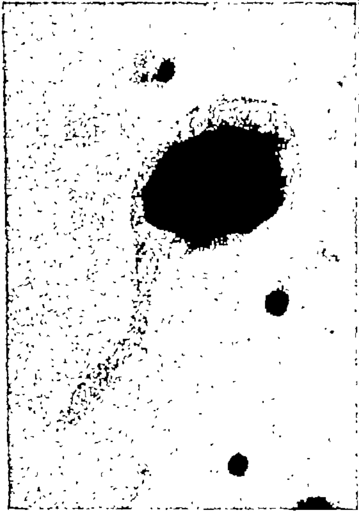


Fig. 3.

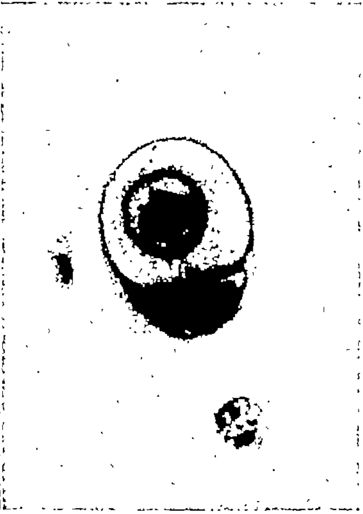


Fig. 4.

Fig. 1.—Cluster of malignant cells (×320).
Fig. 2.—Single hyperkeratinized carcinoma cell (×1,575).
Fig. 3.—Tadpole cell (×1,575).
Fig. 4.—Bird's-eye formation (×1,440).

RESULTS

Our results in general do not compare too favorably with some of the enthusiastic data published by other investigators, perhaps by virtue of our extreme conservatism in making positive reports (Table I). Only when a substantial number of cells adhering strictly to the established criteria was seen *was a positive report made.*

TABLE I. ACCURACY OF THE METHOD REPORTED BY OTHER INVESTIGATORS

AUTHOR	NO. OF CASES IN SERIES	NO. OF CASES BRONCH. CA.	CYT. POS.		FALSE NEG.		FALSE POS.
			(NO.)	(%)	(NO.)	(%)	(NO.)
Wandall[8]	193	100	84	84	16	16	6
Herbut and Clerf[10]	?	57	47	82.4	10	17.6	?
McKay and associates[11]	170	54	40	74	14	26	3
Farber[12]	500	71	57	80	14	20	?

Of the 270 cases studied at Barnes Hospital, 100 showed primary bronchiogenic carcinoma. In 81 instances the clinical diagnosis was histologically confirmed by the following means:

Pneumonectomy	25
Exploratory thoracotomy	33
Bronchoscopic biopsy alone	12
Aspiration biopsy	4
Necropsy	2
Lymph node biopsy	5
	81

In the remaining 19 cases a clinical diagnosis of bronchiogenic carcinoma was made on the basis of symptoms, physical and x-ray findings, and distant metastases in one case to the ulna, and in another to the brain. From the 100 primary carcinomas, positive smears were obtained in 61 cases, suspicious in 8 cases, and negative findings were reported in 31 (see Table II). A positive bronchoscopic biopsy was obtained in only 38 per cent of the cases. Smear and biopsy were positive simultaneously in 25 instances, smear was positive alone in 36, while biopsy was positive alone in 13 cases. Thus when both methods were used to complement each other a correct preoperative microscopic diagnosis was made in 74 per cent of the cases. This finding is almost identical to that reported by Liebow and associates[13] whose positive preoperative microscopic diagnoses increased from the 38 per cent available from bronchoscopic biopsies alone to 76 per cent by use of the smear.

TABLE II. CYTOLOGIC REPORT OF BARNES HOSPITAL CASES

CONFIRMED BY	TOTAL	POSITIVE	SUSPICIOUS	NEGATIVE
Pneumonectomy	25	16	0	9
Thoracotomy	33	20	4	9
Bronchoscopic biopsy	12	7	0	5
Aspiration biopsy	4	2	0	2
Necropsy	2	1	1	0
Biopsy of nodes	5	2	1	2
Clinically positive	19	13	2	4
Total	100	61	8	31

DISCUSSION

One of our first concerns in the study of the cytologic method was whether sputa or bronchial secretions were more satisfactory for the purpose of making and interpreting the slides. Herbut and Clerf,[10] as well as McKay and associates,[11] reported that better results were obtained by use of bronchial aspirations collected at bronchoscopy than by expectorate, because these secretions were less dilute and devoid of cells from the mouth or pharynx which might cause confusion. Theoretically the aspirated secretions should be more likely to contain tumor cells since the material is taken directly from the area of the neoplasm, while sputum is an accumulation of secretions from many areas. Comparing the relative merits of sputa and bronchial secretions, Liebow and associates[13] found that with only one sputum specimen or one sample of bronchial secretion the latter were positive in 30 per cent of the cases but sputa were positive in only 15 per cent. They admitted, however, that the incidence of false positives was also twice as great, possibly explained on the basis that in smears of bronchial aspirations the ciliated columnar cells are often clumped into such dense groups that the cilia are obscured, the nuclei appear hyperchromatic and because they are active show tendencies toward more coarsely granular, almost clumped, rather than finely granular chromatin. This makes the interpretation of the smears difficult, at times even hazardous.

With regard to the preparation of the slides, we find sputum definitely easier to handle. It is not only more convenient to obtain the initial specimen but repeat specimens are more readily available. It does not seem justified to subject the patient to several bronchoscopies just to obtain bronchial washings. Although it has been stated that patients with early lesions do not have a productive cough, we have been able to isolate malignant cells from scanty specimens which qualitatively appeared to consist mainly of saliva. On the other hand the bronchial washing from these same cases is usually clear saline which on centrifugation deposits only a thin film at the bottom of the test tube inadequate for the preparation of slides. The fact that sputum alone can be perfectly satisfactory is evidenced by the remarkable success of Wandall[8] in demonstrating tumor cells in 84 per cent of his cases.

THE OPERABLE CASES

In checking the literature we found Wandall[8] to be the only one who concerned himself with determining whether cytologic examination is more successful in operable or inoperable cases. He reported that neoplastic cells were seen in operable cases (83.3 per cent) almost equally as frequently as they were found in inoperable cases (83.9 per cent). In our series greater success was achieved by studying the material from operable cases. In the group of 25 patients who had pneumonectomy a positive cytologic diagnosis was made in 16 (64 per cent), while it was possible in only 45 (60 per cent) of the inoperable cases. The figures speak well for the method as an adjunct in the diagnosis of early bronchiogenic carcinomas, especially when bronchoscopic biopsy was positive in only 9 (36 per cent) of the resectable cases (Table III).

The fact that such a small number of cases produced positive biopsies may be explained on the basis that 16 lesions were located in the upper lobe and consequently lay beyond the reach of the bronchoscope.

TABLE III. CYTOLOGIC AND BRONCHOSCOPIC FINDINGS IN TWENTY-FIVE PNEUMONECTOMIES

LOCATION OF TUMOR		BRONCHIAL FINDINGS				BRONCHIAL BIOPSY			CYTOLOGY	
		TUMOR VIS.	IND. EVID.	NEG.	NO BRONCH.	POS.	NEG.	NONE	POS.	NEG.
Upper lobe	(16)	3	4	7	2	4	4	8	11	5
Middle or Lower	(9)	6	2	0	1	5	3	1	5	4
Total	(25)	9	6	7	3	9	7	9	16	9

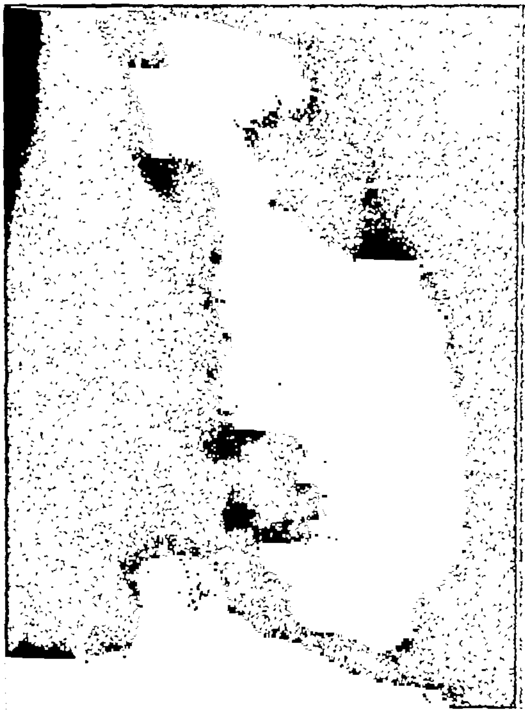


Fig. 5.

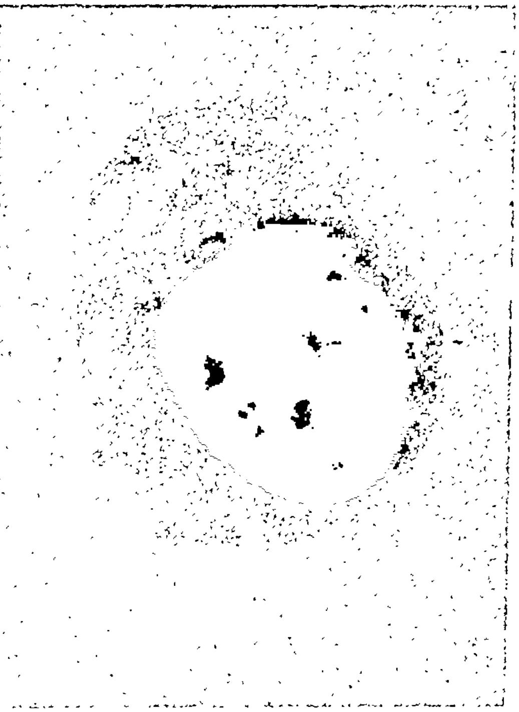


Fig. 6.

Fig. 5 (Patient C. H.).—Roentgenogram showing mass in left upper lobe with central cavitation.

Fig. 6.—Malignant cell representative of those found in the sputum (×2400).

Since only nine positive biopsies were obtained, frozen section was resorted to eight times. In two cases it confirmed the positive cytologic diagnosis; in one case it confirmed the bronchoscopic biopsy; in one case it confirmed both smear and bronchoscopic biopsy; in four cases it was the only histologically positive diagnosis. Pulmonary resection was carried out in four cases on cytologic diagnosis alone and in all of these tumor was found to be present. Two of these four cases are of particular interest and are abstracted briefly below:

CASE 1.—C. H. (Record No. 161727), a 40-year-old white man, stated that eleven days before entry in July, 1948, he had a routine chest roentgenogram which was said to reveal a shadow in the left upper lobe. The patient denied all symptoms save a mild cough which had become worse three weeks prior to entry. Physical and laboratory examinations were essentially negative. Bronchoscopy revealed no abnormalities in the areas visualized and no biopsy was taken. Cytologic examination of sputa was positive for malignant cells. Pneumonectomy without frozen section was carried out. Examination of the gross specimen revealed a 5- to 6-cm. tumor mass in the left upper lobe which was identified microscopically as epidermoid carcinoma, grade 2. Twenty-one regional lymph nodes were free from tumor (Figs. 5, 6, 7, and 8).

CASE 2.—N. P. (Record No. 166787), a 75-year-old white woman, entered the hospital in December, 1948, with a history of left chest pain associated in onset with an upper respiratory tract infection of six months' duration. Chest roentgenogram by private physician showed a shadow in the left upper lobe. Physicial examination was negative. Bronchoscopy showed slight restriction of the left mainstem bronchus in its lateral motion. Cytologic examination of sputum was positive for malignant cells. Pneumonectomy without frozen section was carried out. Grossly there was a fleshy, firm, pale yellow lesion approximately 6 to 7 cm. in greatest diameter occupying the major portion of the apical segment of the upper lobe. Microscopically the tumor was composed of lymphocytic cells larger than ordinary lymphocytes, probably representing lymphoblasts. This lesion was thought to be a primary lymphosarcoma of the left upper lobe. Eleven regional lymph nodes showed no evidence of tumor (Figs. 9, 10, 11, and 12).

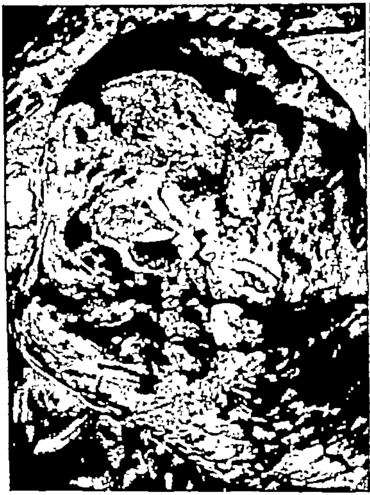


Fig. 7.

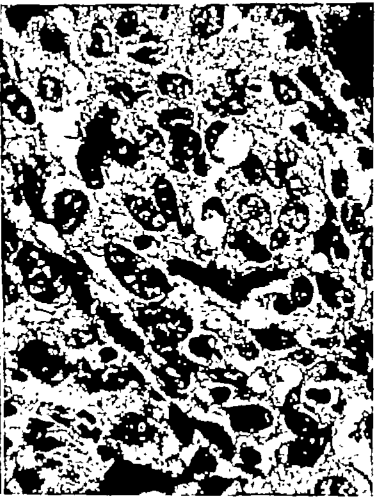


Fig. 8.

Fig. 7.—Gross specimen.
Fig. 8.—Photomicrograph of tumor cells as seen in tissue section.

THE INOPERABLE GROUP

The inoperable group showed a picture somewhat different from that of the resectable group. Cytologic diagnosis was positive in only 45 (60 per cent) of the 75 cases, and an additional 8 were reported as suspicious. While the percentage of accurate cytologic diagnoses dropped, the incidence of positive bronchoscopic biopsies rose to 40 per cent. This difference was undoubtedly due to the fact that in the nonresectable cases larger tumors involving areas more accessible to the bronchoscope were encountered. These tumors tend to be more necrotic often causing the patient to cough up mucopurulent material in which it is virtually impossible to detect well-preserved, recognizable tumor cells. This group is apparently responsible for a significant portion of those cases in which the smear is reported negative although the patient is subsequently found to harbor a malignant neoplasm of the lung.

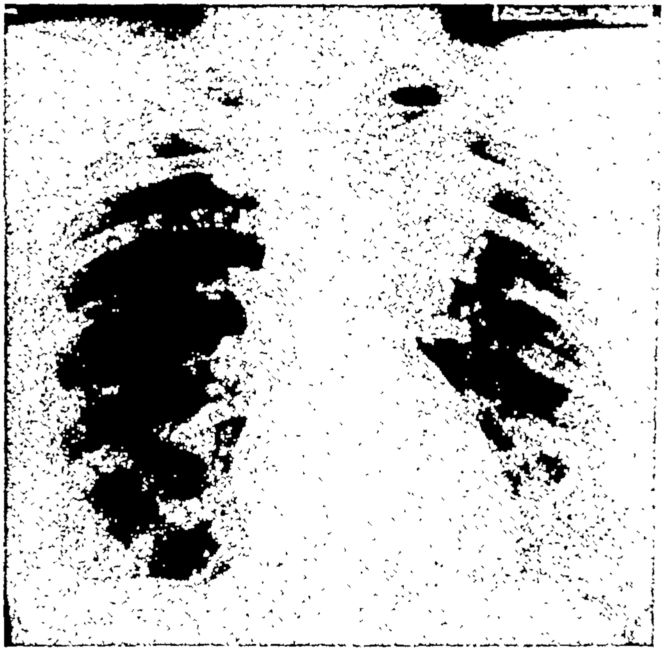


Fig. 9 (Patient N. P.).—Roentgenogram showing left upper lobe infiltration.

LIMITATIONS

Certain limitations inherent in the method must be recognized. A diagnostic test of this sort can, for instance, provide no information regarding either the exact location or origin of the tumor. Thus it was the experience of Woolner and McDonald[14] that in four cases in which a positive sputum had been reported, the carcinoma was demonstrated to be in the larynx, trachea or esophagus rather than in the lung.

Some investigators report being able to recognize the type of neoplasm by study of the exfoliated cells. Herbut and Clerf,[15] Woolner and McDonald,[16] the workers at Vincent Memorial Hospital,[17] and Farber and associates[18] have correlated (1) large, atypical single cells which adhere to the criteria of malignancy and show a hyperkeratinized cytoplasm with squamous-cell carcinoma, (2) small, rather uniform, hyperchromatic, anaplastic cells with oat-cell carcinoma, and (3) malignant cells whose cytoplasm contains large vacuoles with adenocarcinoma. These correlations are based on the finding of such cell forms in contact smears made directly from the tumor. It is our opinion that although such a correlation is possible in contact smears, it does not hold true for exfoliated cells in the sputum since these are subject to degenerative changes. Although it has been our experience that the single hyperkeratinized cell occurs exclusively in epidermoid carcinoma, we have found that cells with large vacuoles are not specifically from adenocarcinoma nor do the small anaplastic cells represent only oat-cell carcinoma. Fig. 13, for instance, shows a group of vacuolated cells which we obtained from an epidermoid carcinoma, the vacuolization apparently being degenerative change. It was interesting to note in this case that all the slides showed only clusters of this type. Fig. 14 shows small anaplastic cells found in a lymphosarcoma rather than oat-cell carcinoma.

FIG. 10.—Gross specimen.

In view of these findings, we prefer not to attempt to recognize from exfoliated cells the type of neoplasm present but report only the presence or absence of malignant cells.

False Negatives.—The so-called false negative incidence—a series of negative smears from a patient with tumor—seems unavoidable. This incidence has been reported to be as low as 16 per cent[8] and as high as 57.2 per cent,[13] though generally it lies between 25 and 35 per cent. Its existence can be based on three factors: first, inadequate specimens; second, failure to transfer the tumor cells from the specimen to the slides; third, failure to identify tumor cells present in the smear. In our study we failed to demonstrate malignant cells in 31 patients with known bronchiogenic carcinoma. Quite a few of these can be attributed to the preceding reasons; for instance, we received many night cups containing a 6 to 8 hour collection of expectorate which as a rule contained in addition to mucoid secretions and saliva, foreign substances such as bits of Kleenex, cigarette butts and cigarette ashes, particles of food, and Lipiodol from bronchography done earlier that morning. It was impossible to select suspicious areas in many cases. Vast growths of bacteria also accumulated in material left standing in warm temperatures causing marked degeneration of the cells.

Obviously little can be done to increase the number of instances in which particles of tumor are transferred from the sputum specimen to the slide. Even when suspicious, blood-streaked areas are smeared, they may or may not contain malignant cells. The job of isolating tumor cells can be facilitated by the collection of sputum after a deep cough and having the patient expectorate only once rather than allowing a collection of material. This is particularly applicable when the patient has a cough highly productive of mucopurulent material.

We feel that the number of false negatives can be reduced not only by the cooperation of the Nursing Service and bronchoscopists in supplying adequate fresh specimens, but also by the study of more than one specimen per case if the initial specimen is negative. Of the 61 cases in which a positive cytologic diagnosis was made, positive smears were obtained from the first specimen in only 32 instances; in 21 cases a second specimen was required before a positive report could be made, and it was necessary to study even a larger number of specimens in 8 other cases. In the false negative group of 31 cases, only one specimen was received for study in 8 cases and two consecutive specimens in 10. Thus in over one-half of this group we failed to receive the three specimens we feel are necessary for greatest possible accuracy.

The possibility that the examiner may overlook malignant cells on a slide exists, of course. Although careful screening of the slides can reduce the number of cases in which malignant cells are missed, there will always be cases in which even the most thorough examiner will overlook neoplastic cells. The margin of error is undoubtedly due to the extreme conservatism necessary in making positive reports. In our laboratory a positive diagnosis is made only on cells entirely compatible with the criteria established for the recognition of tumor cells. It may well be that cells which appear atypical but which do not expressly adhere to these criteria are passed over by the examiner. At one point in our study, after we had made an unusually large number of false negative reports we allowed ourselves to be more lax with the result that two false positive diagnoses were made. We now adhere strictly to our former conservatism.

False Positive.—This source of error can be greatly reduced as the examiner gains experience in the interpretation of smears, though there is some question in our minds whether or not it can be eliminated altogether. Certain lesions of an inflammatory nature such as tuberculosis, bronchiectasis, lipoid pneumonia, lung abscess, or chronic bronchitis seem to produce cell changes which morphologically mimic the characteristics ascribed to carcinoma. The

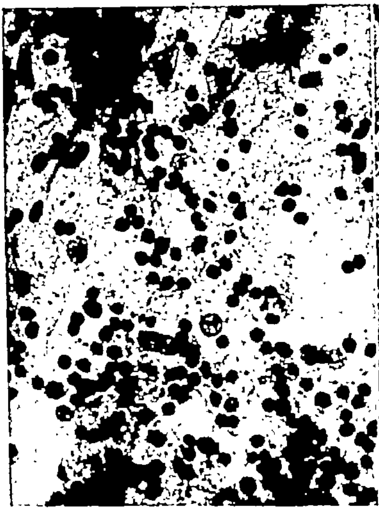


Fig. 11.

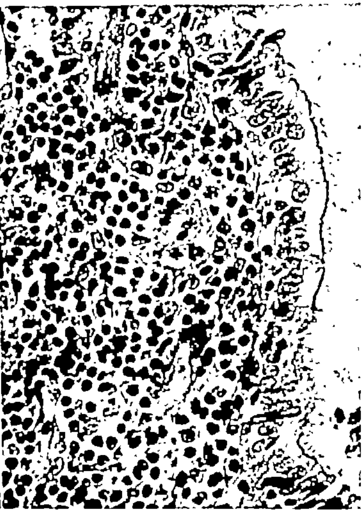


Fig. 12.

Fig. 11.—Cells representative of those seen in sputum and reported malignant (×400).
Fig. 12.—Photomicrograph of tumor cells as seen in tissue section (×400).

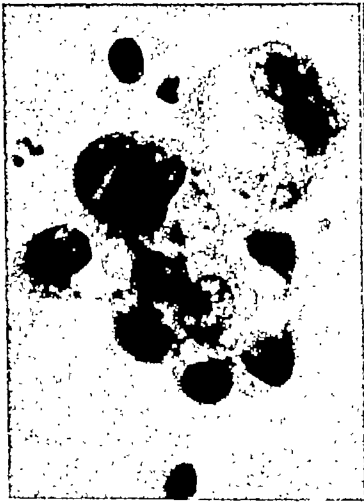


Fig. 13.

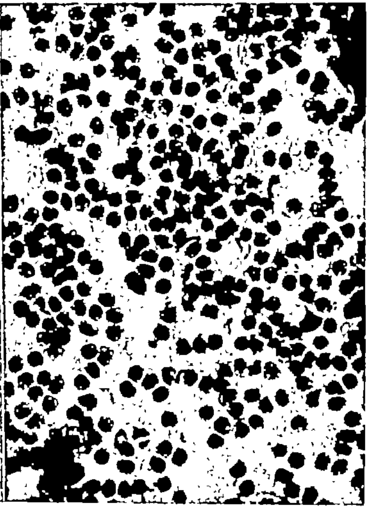


Fig. 14.

Fig. 13.—Vacuolated cells from an epidermoid rather than an adenocarcinoma (×950).
Fig. 14.—Small anaplastic appearing cells from lymphosarcoma rather than oat-cell carcinoma (×400).

Fig. 15.

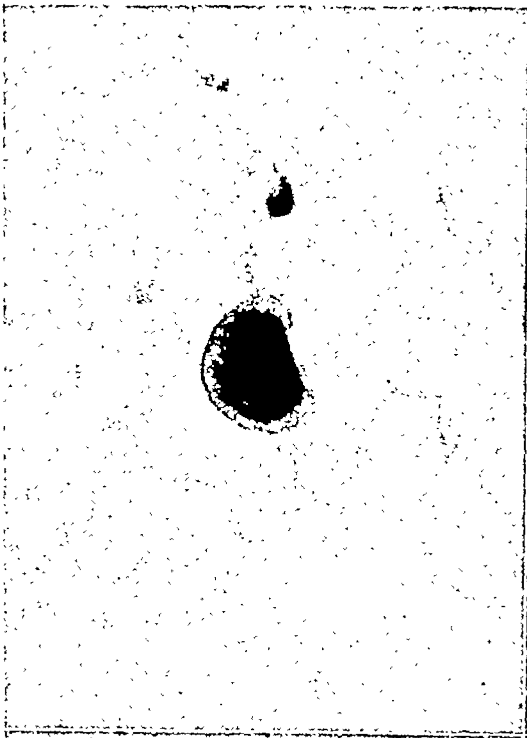
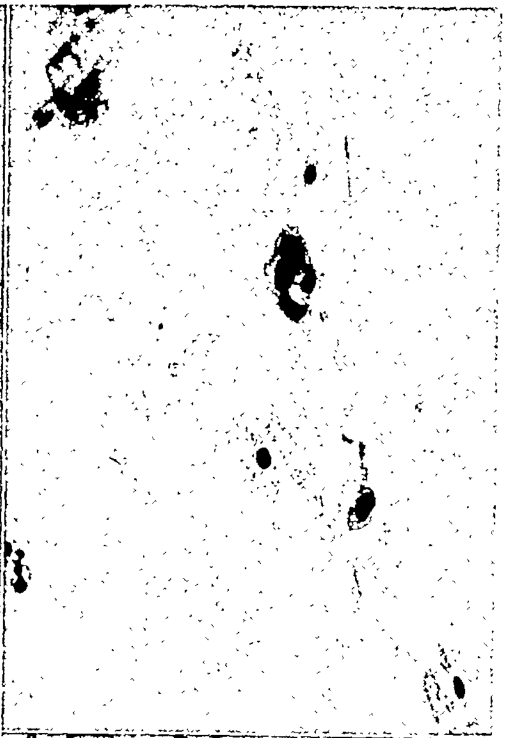


Fig. 16.



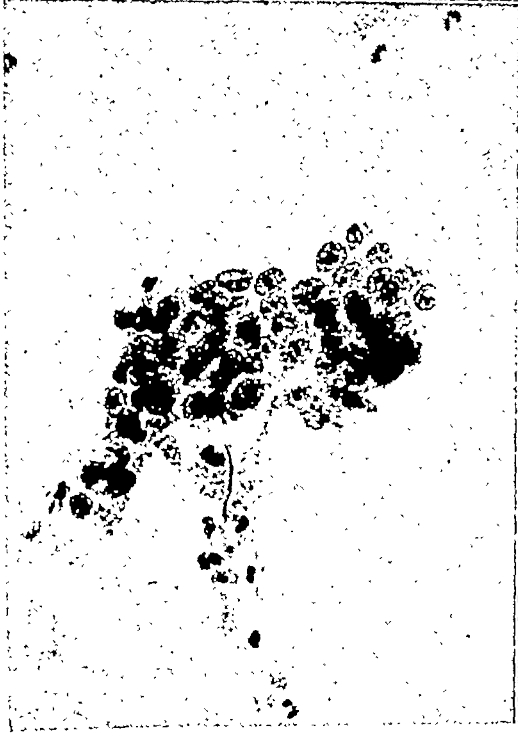


Fig. 17.

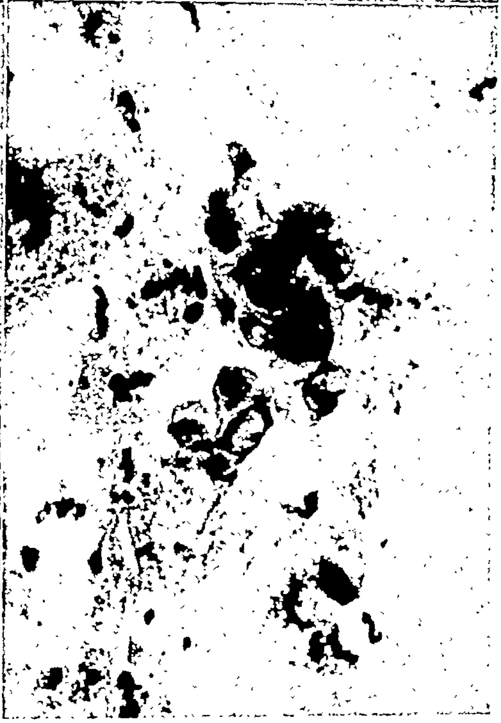


Fig. 18.

Fig. 15.—Malignant appearing cell from a case of chronic bronchitis (×600).

Fig. 16.—A group of such cells from the same specimen as Fig. 15 (×280).

Fig. 17.—A cluster of cells with multiple nucleoli and clumped chromatin from a case of lung abscess (×400).

Fig. 18.—Atypical, deceptive cells from a case of tuberculosis (×600).

recognition that these misleading cells occur is corroborated by other investigators who also made false positive diagnoses on finding such cells in expectorate or bronchial aspirations (Table IV). Of our four false positives, two were made in cases of tuberculosis, one in a case of lipoid pneumonia with associated bronchiectasis, and the fourth remained undiagnosed even after exploratory thoracotomy. On review of these four cases we feel that mistakes in the identification of suspicious cells, illustrated by Figs. 15, 16, 17, and 18, were made in three instances, but that in the fourth case, a true false positive was encountered. Because this last case was particularly instructive, it will be abstracted below.

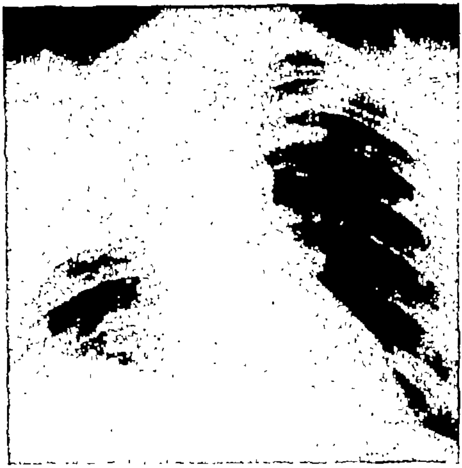


Fig. 19 (Patient W. B.).—Chest roentgenogram showing absent right upper lobe and infiltration of the lower lobe.

CASE 3.—W. B. (Record No. 133308), a 37-year-old white man, was first admitted to Barnes Hospital in March, 1947, at which time a diagnosis of bronchiectasis and bronchostenosis of the right upper and right middle lobe, cause undetermined, was made. The patient improved markedly following bronchoscopy and was discharged. Four months later, the patient complaining of acute illness characterized by severe cough, high fever, and hemoptysis, was readmitted. Bronchoscopy revealed the bronchi of the upper and middle lobes to be almost completely occluded by a diffuse inflammatory reaction with much edema. A right upper lobectomy was carried out one month later. Pathologic diagnosis: bronchiectasis and bronchial lithiasis. On the next admission in August, 1947, a diagnosis of pulmonary tuberculosis was made. In September, 1947, the next admission, the diagnosis was ?empyema, ?lung abscess, broncholithiasis, and pulmonary tuberculosis, right. In August, 1948, the patient was again admitted for recurrence of symptoms. Chest x-ray examination revealed a pulmonary infiltration of the remaining portion of the right lung thought possibly to be tuberculosis. Bronchoscopy revealed nothing unusual in the areas visualized. Review of the slides by the present pathologist showed no tuberculosis. In view of the patient's respiratory distress, a right middle and lower lobectomy was done. Pathologic diagnosis: lipoid pneumonia with associated bronchiectasis. Although many sections were made, no tumor could be found (Figs. 19, 20, 21, and 22).

The cells seen in this case displayed rather obviously the criteria of malignancy. We certainly would be inclined to make a positive diagnosis should we encounter cells of this appearance again. Cases such as this make it apparent that occasional false positives are unavoidable and must be considered an inherent limitation of the cytologic method of diagnosis.

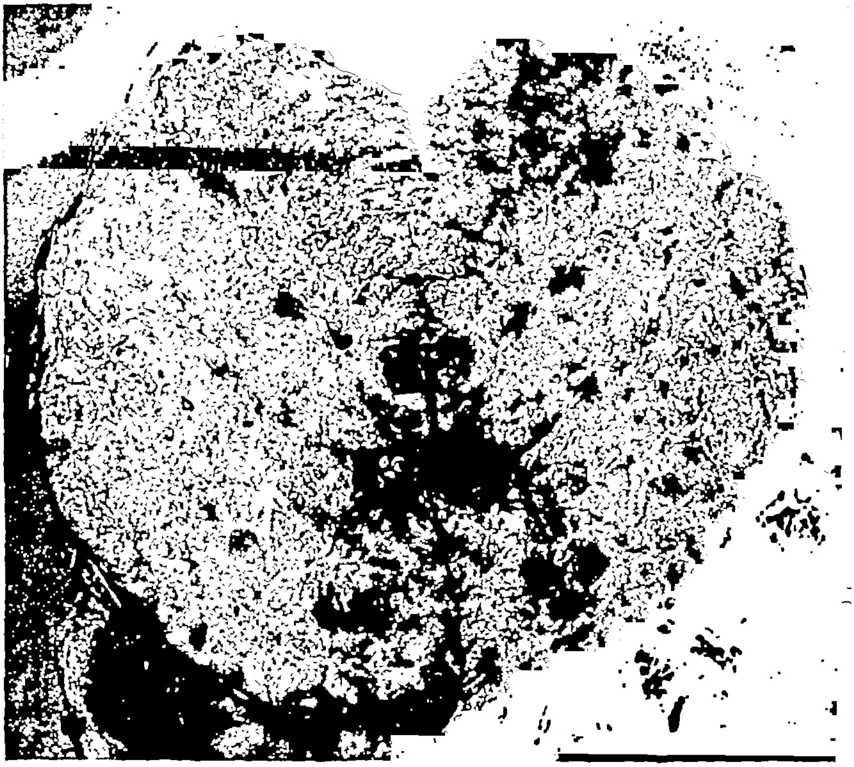


Fig. 20.—Gross specimen of the lower lobe demonstrating circumscribed area of lipoid pneumonia.

TABLE IV. CONDITIONS IN WHICH FALSE POSITIVES WERE OBTAINED

AUTHOR	NO. CASES IN SERIES	NO. OF CANCERS	NO. FALSE POSITIVES	DISEASES IN WHICH FALSE POSITIVES OCCURRED
Wandall[8]	193	100	7	Benign bronchial growth; chronic pneumonia; pulmonary tuberculosis
McKay and co-workers[11]	170	54	3	Pulmonary tuberculosis; bronchial polyp
Liebow and co-workers[13]	131	63	3	Bronchiectasis
Woolner and McDonald[19]	588	147	4	Chronic pneumonitis in one case; others are not given in the report
Watson and co-workers[20]	400	236	4	Bronchiectasis
Our series	270	100	4	Lipoid pneumonia; pulmonary tuberculosis; bronchiectasis

INDICATIONS FOR USE OF THE METHOD

We have come to recognize that the cytologic examination of expectorate does not lend itself for screening large portions of population. The time and cost involved in such an undertaking would be prohibitive, for it takes nearly an hour to interpret four negative slides and the minimum cost per specimen is between $3.00 and $5.00. A further drawback in the use of this method for screening is the relatively high false negative incidence. A single negative specimen means nothing in respect to the presence or absence of bronchiogenic carcinoma in a given patient and the yield of positives would be extremely small. The collection of fresh, adequate material presents another problem, since normal, healthy individuals do not have secretions in their bronchi which can be coughed up.

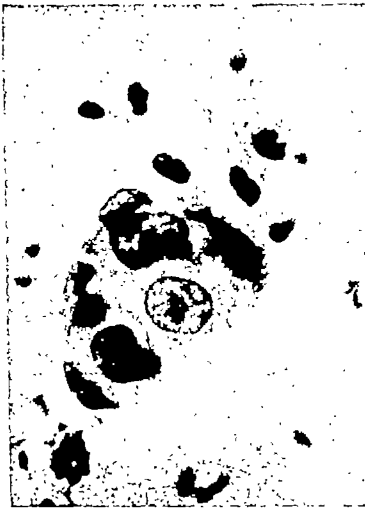


Fig. 21.

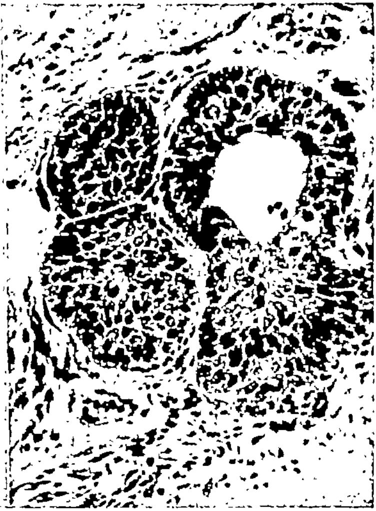


Fig. 22.

Fig. 21.—Cluster of cells interpreted to represent malignant cells. Several such clusters were seen in the sputum (×1,080).

Fig. 22.—Tissue section showing squamous metaplasia which possibly represents the source of the atypical cells found in the sputum (×400).

There are cases, however, in which the cytologic method can be most useful, especially is it valuable as an adjunct in the diagnosis of the disease in patients with minimal symptoms, indeterminate physical and x-ray findings and on whom other preoperative histologic evidence is unavailable. In our study the method proved its worth in the 18 cases with negative bronchoscopy in which the positive smear was the only histologic evidence of malignant pulmonary neoplasm before thoracotomy.

CONCLUSIONS

1. In view of the very deceptive and minimal signs and symptoms of early bronchiogenic carcinoma and the low yield of positive bronchoscopic biopsies, additional adjuncts in diagnosis are imperative. The cytologic diagnosis is discussed as serving such a purpose.

2. Our report is based on the study of expectorate and bronchial aspirations from 270 patients admitted to the hospital for diagnosis and treatment of lung disease. Included in this group are 100 cases of bronchiogenic carcinoma from which positive smears were available in 61 per cent. A higher degree of accuracy was obtained in the operable cases: nearly two-thirds of the resectable cases gave positive cytologic evidence of the presence of carcinoma. It became apparent that the early lesions commonly had a low percentage of positive bronchoscopic biopsies but tended to yield a larger number of cytologic diagnoses.

3. We found that sputum studies were more reliable than bronchial aspirations and afforded us the chance of studying more than one specimen per case.

4. Certain limitations inherent in the method were recognized. Most prominent of these was the incidence of false negatives and false positives.

5. A false negative incidence of 31 per cent occurred in our series, but reduction of the number of false negative reports may be achieved in the following ways:

   a. Only fresh sputum adequate but not copious in quantity should be used for study. Expectorate collected over a period of hours does not constitute a satisfactory specimen.

   b. Painstaking care must be exercised in selecting whitish areas or blood flecks from the specimen for preparation of the slides.

   c. The study of at least three consecutive specimens per case is required for greatest accuracy.

6. If adequate specimens are obtained, the number of correct positive reports could be raised to 75 or 80 per cent for all cases, and for the resectable cases it could be raised to 85 per cent.

7. Four false positives are reported. On review of the slides, we feel that mistakes in interpretation of suspicious cells were made in three instances. In the fourth, cells were seen which even after long experience could still be mistaken for carcinoma.

8. The identification of the type of tumor from exfoliated cells is not reliable.

9. In our opinion this method of diagnosis should be restricted to cases with signs and symptoms of tumor but without an available positive histologic diagnosis. It is not practical as a screening procedure since too much time and expense are involved. The cytologic diagnosis added to clinical and x-ray evidence in the absence of a positive biopsy may be sufficient to sway the balance toward pneumonectomy. We feel that certain cell patterns are practically diagnostic, but that frozen section should be done at thoracotomy when bronchoscopic biopsy is negative even if the cytologic diagnosis has been positive in order to reduce error to practically zero.

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EOSINOPHILIC GRANULOMA OF RIB

WITH A CASE REPORT

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INTRODUCTION

EOSINOPHILIC granuloma of bone is an uncommon lesion affecting more commonly the membranous bones, either in monostotic or polystotic form. Those with involvement of the rib may be seen by the thoracic surgeon. It must therefore be considered in the differential diagnosis of lesions of the ribs. The purpose of this paper is to briefly review the salient features of the malady and to report a case studied and treated by the Department of Thoracic Surgery at the Mercy Hospital.

The most complete early historical description of such bone lesions was by Fraser[1] in 1935. He ascribed the name lipoid granulomatosis of bone to this condition. Later, Lichtenstein and Jaffe[2] reported on cases of solitary bone lesions which pursue a benign clinical course. Their cases resembled histologically those lesions described by Fraser. They presented the clinical features of the disease which they called eosinophilic granuloma of bone. These investigators offered no definite etiological explanation for the condition. It was not until the studies of Farber[3] that the resemblance of the lesions of eosinophilic granuloma of bone to the essential lesions of Schüller-Christian disease was suggested. Thannhauser[4] asserted that this malady did not merit consideration as a specific entity. Jaffe and Lichtenstein take the contrary position that eosinophilic granuloma of bone definitely differs from Schüller-Christian's disease from a clinical, therapeutic, and prognostic standpoint. Although there remains much to be learned concerning the relationship between this condition and the diseases of lipoid metabolism and the reticuloendothelial system, the majority of opinion suggests that eosinophillic granuloma of bone is a monosymptomatic form of Schüller-Christian's disease. Cases have been reported showing various gradations of relationship between the two diseases.[5, 6]

Clinical Picture.—Eosinophilic granuloma is seen most frequently in children and young adults, being extremely rare after the age of 25 years. Men appear to be afflicted more commonly than women.[7] There has been no demonstration of a heredito-familial tendency in the disease. The symptomatology varies, but outstanding in the history is the quite sudden onset of swelling, pain, and tenderness over the affected area. Although trauma is believed not to be an etiological factor, trivial trauma, inducing a pathologic fracture in the region of the lesion[8] has been reported in some patients, preceding the clinical appearance of the granuloma. A mild febrile reaction may accompany the onset of the

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disease. Other general symptomatology is lacking, and in the monostotic form visceral involvement is absent. The clinical course is benign and the prognosis good. Simple curettage or mild x-ray therapy have both effected cures. Spontaneous healing of lesions has been reported.[8]

Laboratory Findings.—Examination of the peripheral blood usually reveals a norml blood picture; a slight degree of eosinophilia may be present. Serum calcium, phosphorus, and alkaline phosphatase determinations are within normal limits. These patients are normocholesterolemic although the total blood fat has been reported to be elevated.[3]

Despite the description of a characteristic x-ray appearance, namely, a round or oval osteolytic lesion originating in the medullary cavity with a punched out appearance, nevertheless, the lesion roentgenographically may simulate Ewing's sarcoma of bone, myeloma of bone, reticulum cell sarcoma of bone, Brodie's abscess, metastatic carcinoma, and simple bone cyst. If the lesion is seen early in the course of the disease, a rapid increase in the amount of involvement of the bone may be observed in serial x-ray examinations. Because of the resemblance of this lesion to the many other lesions of bone, the preoperative diagnosis is exceedingly difficult. It is obvious that the final diagnosis depends upon histologic examination.

CASE REPORT

History.—R. C., a 22-year-old white woman, was admitted to Mercy Hospital with the complaint "a painful lump on the left side of my chest." This pain had a sudden onset three weeks prior to admission, when the chest was strapped by the family doctor who diagnosed a strained muscle. When the strapping was removed in a few days, a hard, tender tumor was noticed on the ninth rib in midaxillary line. This swelling rapidly increased in size. There were no systemic complaints, and the patient was otherwise in good health. The past medical history was negative, except for an uncomplicated appendectomy in 1940. The patient required glasses because of being far sighted. There were four siblings, two of which suffered from seasonal pollinosis. Otherwise all are in good health. There was no familial history of similar bone lesions.

Physical Examination.—Physical examination revealed a well-developed, well-nourished, 22-year-old, white woman, who did not appear sick. There was a firm, fixed mass over the ninth rib in the left anterior axillary line. The mass was about the size of a plum and exquisitely tender. No fluctuation of the mass could be determined and the overlying skin showed no discoloration or fixation to the bone. Examination of the other systems was negative. Temperature 98.6° F.; pulse 70 per minute; blood pressure 120/80; respiration 20 per minute.

Laboratory Findings.—Blood count showed 4,800,000 erythrocytes, 6,900 leucocytes. Differential leucocyte count showed segmented neutrophiles 73 per cent; lymphocytes 25 per cent; eosinophiles 1 per cent; basophiles 1 per cent. No abnormal cells were seen.

Urine had specific gravity of 1.025; albumen and sugar were absent; the urinary sediment was not remarkable.

The serum calcium, phosphorus, alkaline phosphatase, and cholesterol were within normal limits. The blood urea nitrogen was 8.4 mg. per cent. Wassermann and Kahn tests were negative.

X-Ray Report.—Anteroposterior Bucky films of the thoracic cage and oblique films of the lower left ribs showed an oval punched out osteolytic lesion of the ninth left rib in the anterior axillary line, which involved the outer cortex and medullary region of the rib (Fig.

1). The pleural side of the rib was intact. Lateral and posteroanterior films of the skull showed no osteolytic lesions or other variations from the normal. No other bone lesion was evident in any of the other ribs, the clavicles, scapulae, or vertebrae.

Clinical Course.—Preoperative diagnosis was uncertain. Ewing's sarcoma of bone and eosinophilic granuloma of bone were considered as the most likely possibilities because of the age of the patient, the history of development of the lesion, and the laboratory and x-ray findings. The patient was operated upon on the third hospital day under local Novocain 1 per cent, pentothal, nitrous oxide, oxygen sequence. Ten centimeters of the left ninth rib in the region of the anterior axillary line were resected with the periosteum, attached intercostal muscles and a segment of the underlying pleura. The adhesion of the parietal pleura to the periosteum on the inner surface of the rib necessitated its resection and opening of the pleural cavity. The observed underlying pleura and lung were otherwise normal in appearance. The postoperative course was uneventful, except for a mild asthmatic episode on the ninth postoperative day. An x-ray film of the chest at this time was negative. There was no evidence of increased perivascular or bronchial pulmonary markings. The patient was discharged from the hospital two weeks after admission, clinically cured.

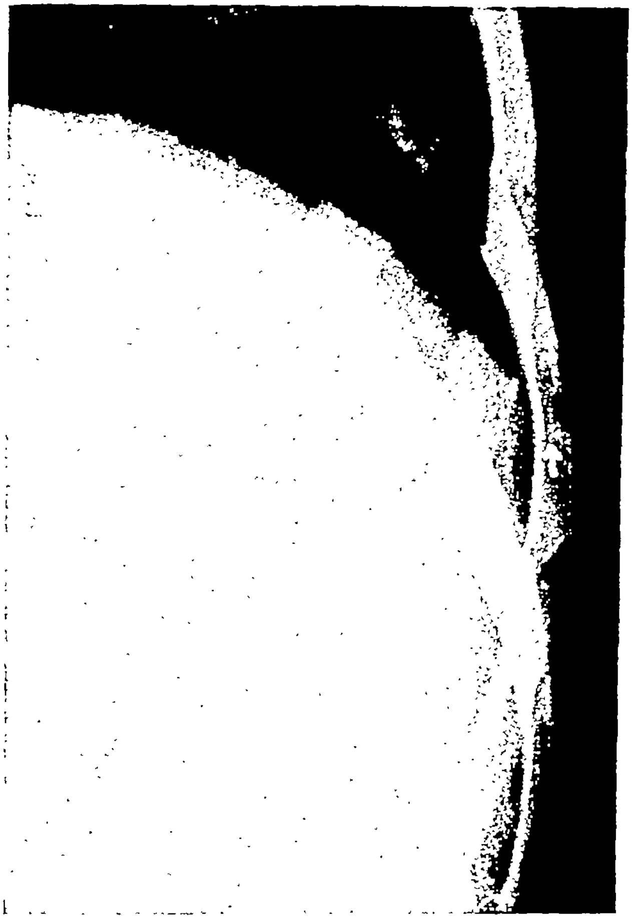

Fig. 1.—Preoperative roentgenogram showing osteolytic lesion in left ninth rib.

Pathologic Report (Abstract).—The cortex of a resected portion of the ninth rib was elevated in a fusiform manner at its midexternal portion. A section through this portion showed expansion of the medullary cavity, which contained a soft grayish-yellow tumor. There was thinning of the overlying cortex (Fig. 2). A Wright's stained smear of the marrow contents showed many eosinophiles, a few large mononuclear phagocytes containing degenerated forms of eosinophiles in their cytoplasm, and the usual marrow cellular constituents. Examination of the hematoxylin and eosin stained paraffin sections of the tumor showed dense collections of eosinophiles and a moderate number of segmented neutrophiles. A few large mononuclear phagocytes were present. These were not vacuolated; their cytoplasm contained debris of degenerating eosinophiles (Fig. 3). The pathological diagnosis was eosinophilic granuloma of rib.

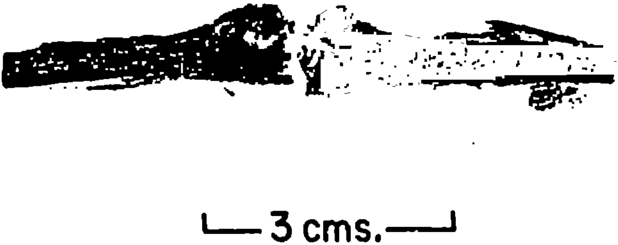

Fig. 2.—Gross specimen of rib showing eosinophilic granuloma with expansion and thinning of overlying cortex.

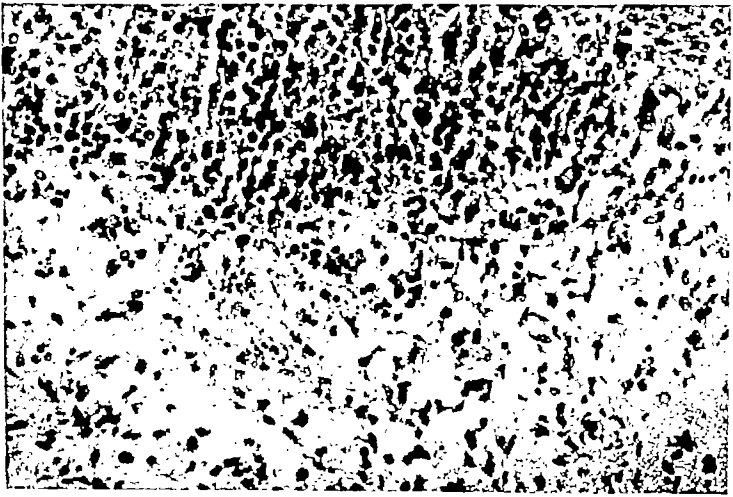

Fig. 3.—Microscopic section of eosinophilic granuloma of bone. Small cells were almost all eosinophiles.

Aspirated sternal marrow smears stained by Wright's method were studied two months after operation. Five hundred cells were counted. Normal maturation and distribution were present. No hyperplasia of the eosinophilic granulocyte series was present. However, examination of five other slides prepared at the same time revealed the presence of an occasional large mononuclear cell, the size of a megalokaryocyte. Its cytoplasm was homogeneous, showing no evidence of lipoid storage, with an indented, moderately dense nucleus. Its larger size and the absence of a nucleolus removed the possibility of this cell being a stem cell of the monocytic series. One similar sized multinucleated cell was seen (Fig. 4). Both types of these cells were identical with those seen in the original Wright stained smear of the marrow adjacent to the lesion in the rib. They are not normal constituents of bone marrow and are considered morphologically compatible with the histiocytes seen in Letterer-Siwe's disease and occasionally early in the Schüller-Christian syndrome.

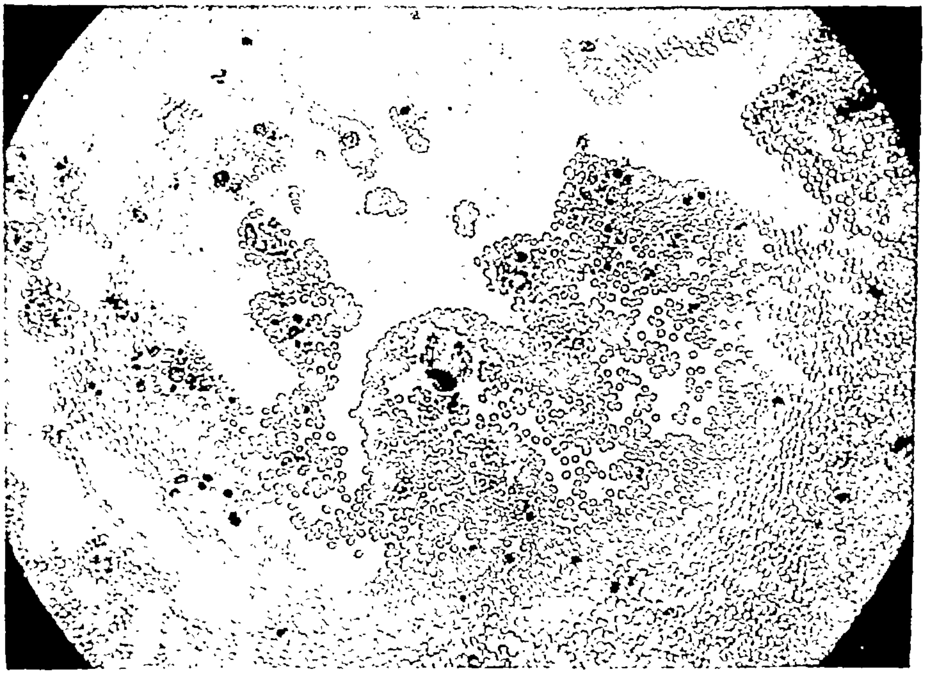

Fig. 4.—Sternal marrow smear showing large multinucleated histiocyte.

DISCUSSION

Although evidence of visceral involvement, characteristic of the Schüller-Christian syndrome, is lacking in this case, nevertheless, the presence of large histiocytes seen in the sternal marrow raises the question of the relationship of this solitary lesion to the generalized disease of lipoid granulomatosis. The lack of available bone marrow studies in previously reported cases over a period of time following recognition of the disease, makes it difficult to ascribe with any certainty the importance of such an observation. It may well represent a characteristic transition of an acute localized phase of a disease which, in its later stages, displays generalized cellular lipoid storage.

CONCLUSIONS

1. The history, clinical and laboratory findings of eosinophilic granuloma of bone are briefly reviewed.

2. A typical case with monostotic rib involvement is presented.

3. The importance of wide surgical excision of the lesion, when accessible, with histologic examination, because of the difficulty of clinical differentiation of this lesion from those of a malignant character, is advised.

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CONGENITAL ABSENCE OF THE PERICARDIUM

A Case Report

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THE first report of congenital deficiency of the pericardium was made by Realdus Columbus in 1559. Since then Moore,[1] in 1925, and Rusby and Sellors,[2] in 1945, have collected a total of eighty cases described in the world's medical literature. Analysis of the early reports in this collective review has cast doubt on several cases which are believed to have been erroneously classified, their true nature being probably that of adhesive pericarditis. Until recently this condition was discovered only as an accidental finding on necropsy examination but lately surgeons have described pericardial defects as an operative finding and a method has been proposed for its clinical recognition.[3, 4] It is interesting to note that in the Abbot monographs considerable space has been devoted to pericardial defects while Taussig makes no refence to this in her textbook on cardiovascular anomalies.

CASE REPORT

The present report deals with a case of congenital absence of the pericardium. Recognition of this anomaly was made in the anatomy laboratory during the course of other studies on the cardiovascular system. The subject was a 12-day-old infant who died of cerebral injury incurred at delivery and who during life gave no symptoms suggestive of cardiac or respiratory pathology. The results of the pertinent findings have been outlined in the following description.

On examination of the thoracic cavity a complete absence of the pericardium was noted. The heart was globular in shape, maintained its normal mediastinal position, and occupied a common serous cavity with the left lung. Except for fixation by the inferior vena cava the distal part of the heart was perfectly free and unattached to the diaphragm. There were no cardiopulmonary adhesions. The heart was covered by epicardium but was completely devoid of pericardium except for a rudimentary fold which extended around its right border. This fold exhibited a free margin in which was contained the left phrenic nerve but was otherwise fibrous and avascular in character. The aorta, the primary pulmonary vessels, and superior vena cava were also without a fibrous investing sheath of pericardium. The sternopericardiac ligaments were absent.

The displacement of the left phrenic nerve from its normal anatomic course in the thorax was of great interest. The left phrenic nerve was dissected in the neck where the usual relationships to the anterior scalene muscle and the cervical plexus were observed. The nerve entered the thorax behind the left innominate vein, crossed in front of the arch of the aorta, and curved sharply to the right joining the rudimentary fold previously described. Its position thereafter was to the right of the midsternal line until it reached the diaphragm at the xiphoid level where it turned sharply to the left and entered the muscle. The nerve was unaccompanied by the pericardiophrenic branch of the internal mammary artery. The course of the right phrenic nerve was normal.

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The heart and its vessels were studied after injection of the vascular system. The anatomic structure of the heart was normal except for an unusual appearing left auricular appendage resembling a clown's cap. The fibrous vestigial band of Marshall was present as a wide band in the region of the posterior aspect of the left auricle. Several other tissue remnants found in the same area were considered to be persistent remnants of tributaries of the obliterated cardinal venous system. The coronary sinus was more dilated than normal. The ductus arteriosus was patent but was in the process of closure. The other vessels were normal.

Abnormalities were sought for in other structures. The left lung showed a normal arrangement of lobes and bronchopulmonary segments. The diaphragm was well developed without defects. Anomalies were not found in the abdominal cavity.

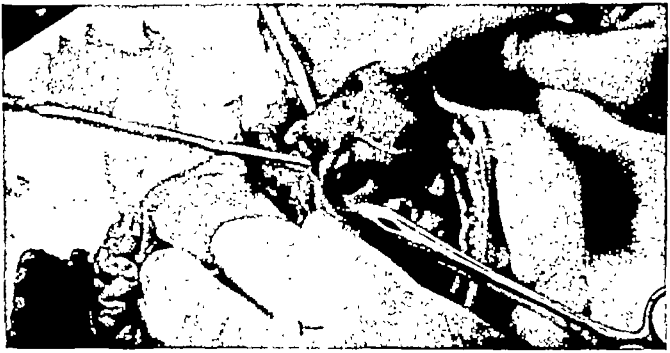

Fig. 1.—Photograph shows heart devoid of pericardium. Clamps have been placed around the rudimentary fold. The heart is freely mobile.

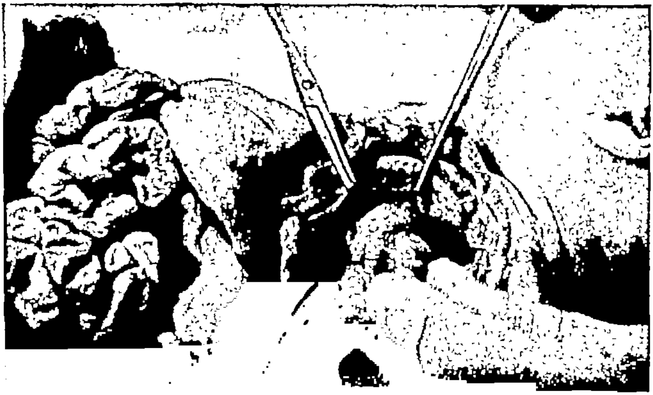

Fig. 2.—Another view showing the rudimentary fold on the right side of heart. In the margin of the fold is contained the left phrenic nerve.

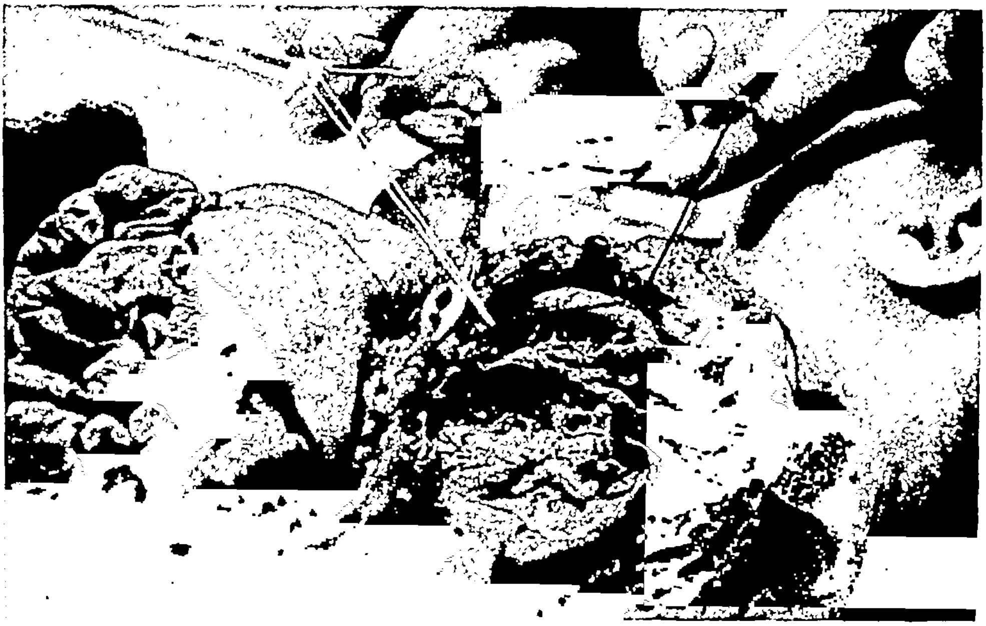

Fig. 3.—Shows the dissection of the left phrenic nerve which is held up by black and white sutures in photograph. Dissection can be followed from the neck as it swings markedly to the right in thorax and then returns to the left to terminate in diaphragm.

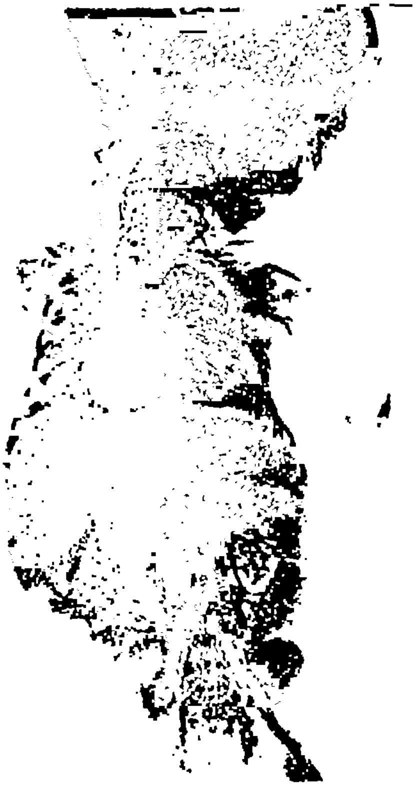

Fig. 4.—Roentgenogram showing position of heart with the cardiovascular tree injected.

The anatomic findings in this case were of developmental significance. The heart and left lung occupied a common cavity embryologically identifiable as the left pleuropericardial space. The anomalous situation of the left phrenic nerve was related to the failure in development of the pleuropericardial membrane. Its displacement to the right was due to the disproportionate growth of the thoracic viscera. The concentration of rudimentary tissue in the region of the auricle seemed to implicate the cardinal venous system in the causation of the pericardial defect.

The pathogenesis of pericardial deficiency has been variously interpreted. Keith claimed that this anomaly was caused by the too rapid growth of the lung bud resulting in an abnormal dilatation of the iter venosum following which the pleuropericardial foramen remained patent.[5] Risel opposed Keith's view suggesting as a cause atresia of the pleuropericardial membrane with failure to join the lung roots.[6] McGarry believed that abnormal tension created by the rotation of the developing liver traumatized the pulmonary ridge from which the membrane developed.[7] Perna and Plaut proposed a vascular atrophy theory based on the hypothesis that premature atrophy of the left duct of Cuvier was causative of a deficiency to the blood supply to the pleuropericardial membrane. The latter theory has been generally accepted because it accounted best for the preponderance of pericardial defects located on the left side.[8]

The functional and physiologic importance of the pericardium always appears to be discussed with congenital deficiency of this structure. Several writers have claimed that the defective pericardium was responsible for elongation and dilatation of the heart due to the lack of pericardial support. Others have stated that this anomaly in itself was without significance, cases being cited in support of this view. All agreed that a heart occupying a common serous cavity with the lung is subjected to the additive factor of respiratory disease. Nevertheless the pericardium has been widely resected clinically and experimentally without creating serious complications.

SUMMARY

A case of congenital absence of the pericardium has been described. Its embryology, the theories of causation, and the significance of pericardial deficiency have been briefly discussed.

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RESECTION VERSUS THORACOPLASTY IN THE TREATMENT OF PULMONARY TUBERCULOSIS

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THE purpose of the paper is to analyze 93 consecutive resections done for pulmonary tuberculosis at the Missouri State Sanatorium at Mount Vernon, Mo., from Jan. 20, 1940 until June 29, 1949. Seventy per cent of these patients have been followed from one to nine years.

We have tried to set up certain indications for resection in pulmonary tuberculosis and to separate them from the indications used for thoracoplasty and other collapse procedures. We recognize that these indications are not always clearly defined but we have carefully reviewed our cases and in our own mind those which we will present for resection versus thoracoplasty are definitive and clear cut. This paper presents our opinions and conclusions at the present time.

The following resections were done:

Left lung	28
Right lung	21
Right upper lobe	13
Right upper and middle lobe combined	5
Right middle and lower lobe combined	4
Right lower lobe	1
Left upper lobe	13
Left lower lobe	6
Right middle lobe	1
Right upper lobe and right lower lobe (apical segment only) combined	1
Total resections:	93

These 93 resections were done upon 93 patients. There were no multiple operations. There were 73 female and 20 male patients. The youngest patient was 9 years old and the oldest was 65 years old. The average age was 30.8 years. Eighty-eight patients were white and 5 were colored. All patients had a positive sputum for acid-fast bacilli before surgery.

Epidural anesthesia has been used at the Missouri State Sanatorium since Oct. 30, 1945. In our opinion this type of anesthesia has played a great part in lowering the incidence of postoperative spreads.

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THE INDICATIONS FOR RESECTION

In this series of 93 patients with proved pulmonary tuberculosis, there are eleven indications for resection. Most of the patients had more than one reason for resection and no attempt is made to give just the primary indication. Often, it is hard to state which indication has preference. Our indications for resection are:

1. Clinical tuberculous bronchiectasis	73
2. "Dead lungs"	36
3. Thoracoplasty failure	22
4. Giant cavitation	20
5. Pneumothorax failure	13
6. Bronchial stenosis	10
7. Lower lobe disease with cavitation	4
8. Bronchopleuro-cutaneous fistula	4
9. Tuberculoma	2
10. Miscellaneous	2
11. Plombage pack failure	1

DISCUSSION OF THE INDICATIONS FOR RESECTION

1. *Clinical Tuberculous Bronchiectasis.*—There is still a great deal of controversy as to when tuberculous bronchiectasis exists. There were 73 patients with this condition in the 93 resection cases. The clinical diagnosis, of course, is made by a Lipiodol bronchogram.

In the majority of cases of pulmonary tuberculosis there is a simultaneous healing and destructive process. Either process may predominate. The healing process is characterized by fibrosis and contraction of the pulmonary parenchyma with a resulting widening deformity of the major bronchi. This condition is what we understand as bronchiectasis. The constant bathing of the bronchi with positive sputum for acid-fast bacilli will often cause endobronchial tuberculosis of these bronchi. Some authorities consider this condition tuberculous bronchiectasis.

If the flow of positive sputum for acid-fast bacilli is checked by a cavity closure, either spontaneously or by some form of collapse therapy, the endobronchial lesion may heal and the tuberculous bronchiectasis is converted to a nonspecific type of bronchiectasis.

As pulmonary tuberculosis usually occurs in the upper lobes and the upper part of the lower lobes, drainage is usually adequate. Therefore, pulmonary resection is not usually indicated in upper lobe bronchiectasis if the sputum is negative for acid-fast bacilli. However, in many instances the endobronchial lesion will not heal after the cavity is closed and a positive sputum for acid-fast bacilli will remain present. In these cases pulmonary resection is the treatment of choice because the tuberculous bronchiectasis cannot be expected to heal by any form of collapse therapy. Thoracoplasty would only collapse the little remaining parenchymal tissue and would have no effect upon the dilated bronchi. Thus, thoracoplasty would only increase the deformity and blockage of the already dilated and obstructed bronchi. Needless to say, streptomycin should always be tried to convert the sputum from positive to negative.

From a pathologic standpoint tuberculous bronchiectasis is present only when there is coexisting bronchial stenosis and tuberculous infection distal to the stenosis with resulting destruction of the muscular and elastic layers of the bronchial wall. There is tubercle formation in the submucosa which may or may not be accompanied by ulceration of the mucosa. Mucosal ulceration depends upon the degree of activity at the time the lesion is seen. This destruction causes weakening and dilatation and what we consider true tuberculous bronchiectasis. Thus, this is a pathologic diagnosis and cannot be made with accuracy before surgery. The late continued dilatation and destruction may in some rare cases be kept progressive by secondary organisms and not be due primarily to the tubercle bacilli.

2. *"Dead Lungs."*—We had 36 patients whom we placed under this classification. Our definition of a "dead lung" is one in which the parenchyma has been completely replaced by fibrosis, cavitation, and bronchiectasis. There usually is a retraction of the lung, mediastinum, and trachea to the affected side. The bronchiectasis is not easily recognized until Lipiodol studies have been done.

This type of case is usually the end result of tuberculous bronchostenosis plus parenchymal disease, or a long-continued, ineffective pneumothorax which may or may not have been abandoned. Patients with this condition have a low vital capacity and are definitely poor operative risks. However, we feel that resection should be done, rather than thoracoplasty, in this type of tuberculosis, although we have performed thoracoplasties as a preparation for resection in extremely toxic, poor-risk patients. The oxygen exchange will not be altered by resection as these dead lungs act only as a continuation of the normal dead space. Unless resection is done in this group, these patients are doomed to a life of incapacity or hospitalization with no chance of cure. Pulmonary resection offers the only chance for a negative sputum for acid-fast bacilli and return to some form of a rehabilitated negative status.

3. *Thoracoplasty Failure.*—Thoracoplasty failure has been our indication for resection in 22 patients. In all patients we waited from six months to one year before considering the thoracoplasty a failure.

There are several sources from which a positive sputum for acid-fast bacilli can originate after a thoracoplasty, and all possibilities should be thoroughly investigated before pulmonary resection is done. Of course, the contralateral side should be excluded as the origin of the positive sputum for acid-fast bacilli.

Laminagrams should be taken of both sides. If residual cavitation is found underneath the thoracoplasty, a revision thoracoplasty can be considered if the thoracoplasty is thought to be mechanically inadequate. If laminagrams fail to detect residual cavitation, the source of the positive sputum for acid-fast bacilli may be from endobronchial tuberculosis. In most instances this condition can be diagnosed by bronchoscopy and treated successfully with streptomycin. Sometimes when the bronchoscopy is noncontributory, the bronchograms will show definite bronchiectasis. At present we consider pulmonary resection indicated in most of those cases. Pulmonary resection is usually indicated when the bronchiectasis is limited to the lower lobes.

We now routinely make preoperative Lipiodol bronchograms on *all* patients being considered for thoracoplasty or resection. The information gained is often more important and influences our decision more than the information gained by bronchoscopy. Several of our earlier patients would have had primary pulmonary resection instead of thoracoplasty if they had been studied with Lipiodol bronchograms first.

To save hospitalization time for the nonconverted thoracoplasty patient, we now make the following routine examinations six months after every thoracoplasty:

1. Laminagrams.
2. Bronchograms.
3. Bronchoscopy.

Treatment is then outlined as previously discussed.

4. *Giant Cavitation.*—There were 20 patients in this group. Cavities measuring more than 4 cm. in diameter are considered as giant cavities. Most of our 20 patients had cavities considerably larger, with some occupying an entire lobe or an entire lung. Most cavities were of the tension type. Pneumothorax is definitely contraindicated in the presence of a giant tension cavity that is accompanied by endobronchial tuberculosis. After analyzing our own cases, we believe that thoracoplasty is a failure in most instances where giant cavitation is present. Resection offers a better hope of cure in those patients who still have a rather poor prognosis. Occasionally, we have recommended thoracoplasty in extremely ill and poor-risk patients as a preliminary to resection.

5. *Pneumothorax Failure.*—We had 13 patients whom we placed in the pneumothorax failure group. The three main reasons for pneumothorax failure are:

1. Bronchostenosis and atelectasis.
2. Lower lobe lesion.
3. Tuberculous bronchiectasis.

All of these reasons are included in our indications for pulmonary resection.

In a few cases of pneumothorax failure the reason will be bronchiectatic cavities or tuberculous bronchiectasis. This type of case does not respond to any form of collapse therapy or to streptomycin because of the scarring and fibrosis of the bronchi. An ordinary film will not show this scarring. The bronchi must be filled with Lipiodol and the upper lobes then show the characteristic multiple beading along the lumen of the bronchi. This multiple beading is characteristic of upper lobe bronchiectasis, as it tends to be a dry type because of adequate drainage in contrast to the wet type seen in lower lobe bronchiectasis. Here again, resection appears to be the treatment of choice as this type of lesion cannot be compressed by thoracoplasty.

6. *Bronchial Stenosis.*—There were 10 patients with bronchial stenosis in the series of the 93 resections. There are two types of bronchial stenosis associated with pulmonary tuberculosis, the ulcerostenotic and the fibrostenotic. The ulcerostenotic type is not necessarily an indication for resection because tuberculous ulceration, which is the cause of the stenosis, may heal with or without stricture, either spontaneously or following streptomycin therapy.

Bronchial stenosis may become particularly prominent after a pneumothorax is started. A lobe or an entire lung becomes atelectatic and assumes the characteristic ground glass appearance, often with definite evidence of cavitation within the atelectatic area. In these cases there is little hope of closing the cavity or cavities, and the pneumothorax should be abandoned. Many times the attempt to re-expand the lung is unsuccessful because of the bronchial stenosis, and resection is then the treatment of choice. Even in the few cases where it is possible to abandon the pneumothorax, it is highly questionable if thoracoplasty, which offers less collapse than the pneumothorax and like the pneumothorax causes kinking of the stenotic bronchi, will result in a sputum conversion of acid-fast bacilli. Again, in this type of case, resection is the treatment of choice.

In the fibrotic type of stenosis we feel that resection is always the treatment of choice, either when dealing with the main bronchi or a lobar branch bronchus providing a positive sputum for acid-fast bacilli is present.

7. *Lower Lobe Disease With Cavitation.*—Nearly every patient with lower lobe lesions will fail to respond to pneumothorax treatment, even when the pneumothorax is combined with phrenicotomy, pneumoperitoneum, or both. The cavity remains open and the sputum continues positive for acid-fast bacilli. In this type of case a thoracoplasty is out of the question, as the inadequate pneumothorax has given a greater degree of collapse than could be expected from a perfect thoracoplasty. In our opinion, one should not temporize in advising resection before a contralateral spread occurs. Pneumothorax would only delay the end result and may be costly and take away the patient's chance of an early successful cure.

8. *Bronchopleuro-Cutaneous Fistula.*—There were 4 patients who had to have pulmonary resection because of a persistent bronchopleuro-cutaneous fistula.

9. *Tuberculoma.*—Two patients had pulmonary resections for tuberculomas.

10. *Miscellaneous.*—Two patients had pulmonary resections for conditions that did not fit into any of the other definite indications. Therefore, these patients are placed in the miscellaneous group.

11. *Plombage Pack Failure.*—There was only one patient that had to have a pulmonary resection because of inadequate plombage.

RESULTS

Mortality.—Nineteen of the 93 resection patients are now known to be dead, an incidence of 20.4 per cent. Seventy-four patients are alive and well at the present time.

The mortality reasons are seen in Table I. This chart also gives the time that death occurred. Cardiorespiratory insufficiency and embolism caused the greatest number of deaths. Bronchopleural-cutaneous fistula and tuberculous empyema were the second greatest causes of death. This condition occurred eight times and was responsible for 42.1 per cent of the deaths. Table II shows the mortality for each type of resection done.

Sputum Conversion.—Sixty-eight patients were sputum negative for acid-fast bacilli at the time of discharge. Thus, 73.1 per cent of the 93 tuberculous resections became negative for acid-fast bacilli. Since 19 of the resection pa-

TABLE I. MORTALITY CHART OF NINETEEN TUBERCULOSIS RESECTION DEATHS

CASE NUMBER	AGE (YEARS)	SEX	RACE	CAUSE OF DEATH	TIME OF DEATH
9,883	26	F.	W.	Generalized toxemia	6 days
14,637	38	F.	W.	Generalized miliary tuberculosis of all organs and bronchopleuro-cutaneous-fistula	2 months
16,809	37	F.	W.	Cerebral embolism	20 hours
17,444	37	F.	C.	Pulmonary insufficiency	17 days after surgery
18,005	43	F.	W.	Cardiorespiratory failure	2 months after resection
16,464	39	M.	W.	Cardiorespiratory failure	7 months after resection
18,187	47	F.	W.	Shock and cardiopulmonary insufficiency	24 hours after resection
18,374	65	F.	W.	Shock (severe cardiac case)	15 minutes after surgery
17,420	63	M.	W.	Tuberculous empyema and bronchopleuro-cutaneous fistula	6 months after resection
16,328	26	F.	W.	Cardiorespiratory failure	7 months after resection
18,416	9	M.	C.	Tuberculous empyema and bronchopleuro-cutaneous fistula	9 months after surgery
17,983	29	F.	W.	Contralateral spread, reactivation, and cardiopulmonary insufficiency	2 months after resection
17,887	36	F.	W.	Acute cor pulmonale and generalized anasarca and contralateral spread	1 month after resection
17,951	45	F.	W.	Bronchopleuro-cutaneous fistula (left hospital against advice)	13 months after resection
19,120	30	M.	W.	Bronchopleuro-cutaneous fistula	4 months after surgery
19,051	27	M.	W.	Cardiorespiratory failure	24 hours after resection
19,053	38	M.	W.	Bronchopleuro-cutaneous fistula and tuberculous empyema	5 months after resection
17,763	38	F.	W.	Bronchopleuro-cutaneous fistula and empyema	7 weeks after resection
18,061	21	F.	W.	Bronchopleuro-cutaneous fistula and empyema	3 months after resection

TABLE II. MORTALITY INCIDENCE FOR EACH TYPE OF RESECTION DONE IN CASES OF PULMONARY TUBERCULOSIS

	PER CENT
1. Right pneumonectomy Eight deaths in 21 resections	38.1
2. Left pneumonectomy Seven deaths in 28 resections	25.8
3. Right upper lobe lobectomy No deaths in 13 resections	0.0
4. Right upper lobe and right middle lobe lobectomy One death in 5 resections	20.0
5. Right middle lobe and right lower lobe lobectomy Four resections with one death	25.0
6. Right lower lobe lobectomy One resection with no deaths	0.0
7. Left upper lobe lobectomy Thirteen resections with no deaths	0.0
8. Left lower lobe lobectomy Six resections with 2 deaths	33.3
9. Right middle lobe lobectomy One resection with no deaths	0.0
10. Right upper lobe and right lower lobe (apical segment) One case with no deaths	0.0

tients have died and 74 patients are living and well, there is an incidence of 91.9 per cent sputum conversions for acid-fast *bacilli in the living patients*, as 68 of the patients were sputum negative for acid-fast bacilli at the time of discharge.

Spreads and Reactivations.—There were 4 patients who had postoperative spreads and 5 patients who had reactivations. Thus, the incidence of spreads was 4.3 per cent and the incidence of reactivations was 5.4 per cent. In our opinion, epidural anesthesia has played an important role in lowering the incidence of postoperative spreads and reactivations.

COMMENT

The mortality in this series may appear rather high. There are several plausible reasons for this condition. Most of the patients who came to resection were extremely poor risks. They had had pulmonary tuberculosis for many years prior to the time when resection was decided upon. Resection was usually done as a last desperate hope of cure and often after other types of treatment had failed. Furthermore, in the early years when resections were done the technique of pulmonary resection had not developed to the present degree, and streptomycin was not available at our institution until 1947.

No doubt better results can be obtained with still further improvement in operative technique and better selection of cases. At present we are evaluating our results with pneumoperitoneum, pneumothorax, thoracoplasty, and other active methods of treatment in different types of pulmonary tuberculosis. In the types of disease where poor results are obtained with the different forms of collapse therapy, we are inclined to do primary resections whenever possible. We hope that our future results with pulmonary resection will save valuable hospitalization time for the patients and reduce further the over-all mortality in all surgical procedures used in the treatment of pulmonary tuberculosis.

SUMMARY

1. Ninety-three consecutive pulmonary resections done for pulmonary tuberculosis at the Missouri State Sanatorium, Mount Vernon, Mo., from Jan. 20, 1940 until June 29, 1949, are analyzed as to types of resection, indications for resection, and end results.

2. The indications for resection are discussed fully so as to show why resection was preferred instead of thoracoplasty or some other type of collapse therapy.

3. The importance of preoperative Lipiodol bronchograms is emphasized.

4. Ninety-one and nine-tenths per cent of the living pulmonary resection patients are now sputum negative for acid-fast bacilli.

5. The incidence of postoperative spreads and reactivations was low and we feel that epidural anesthesia was an important aid in their prevention.

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BLOOD VOLUME AND EXTRACELLULAR FLUID IN TUBERCULOSIS

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RECENT studies on extracellular fluid in chronic infections and cancer[1, 2] have demonstrated a reduced blood volume and an increase of interstitial fluid volume. This report is based on preoperative studies of fifty patients with tuberculosis and twelve healthy adults. Our findings were a reduction in the cell mass, and an increase in interstitial fluid and plasma volume, a decrease in serum albumin, and a decrease in total serum protein in the patients with tuberculosis.

The plasma volumes, extracellular fluid (thiocyanate space) volumes, and hematocrits were done using the method described by Gregersen and Stewart,[3] and from these the cell masses and interstitial fluid volumes were calculated.

The serum albumins and total proteins were done, using the method described by Kingsley.[4]

Twenty-five of the patients with tuberculosis were considered to be in good physical condition, and twenty-five were considered to be in poor condition because of prolonged fever, far-advanced disease, empyema, or excess weight loss.

The averaged cell mass was reduced in the patients with tuberculosis when *compared to the healthy adults. All comparisons were made on the basis of* liters per square meter of body surface area, as this is reported as being a more accurate method of comparison than either cubic centimeters per kilogram of body weight or by total volume.[5] The cell mass in the patients with tuberculosis averaged 990 c.c. per square meter surface area, compared to the average healthy adult of 1,200 cubic centimeters. There was a more marked deficit in cell mass, 900 c.c. per square meter, compared to 990 c.c. when the usual surface area calculated from the patient's optimal weight was used.

Patients who were considered to be in poor condition (Fig. 1) had less cell mass on the average than those considered to be in good condition; 690 c.c. compared to 1,030 c.c. per square meter present surface area.

The 33 patients who had a gain in weight, or loss of less than 20 pounds had 1,700 c.c. cell mass per square meter of surface area. The 17 patients who had lost more than 20 pounds in weight averaged 900 c.c. cell mass per square meter of surface area.

The average total cell mass in the healthy adults was 2,400 c.c., and in the patients with tuberculosis 1,600 c.c.,[1] a deficit of 800 c.c. of cells, or 1,500 to 2,000 c.c. of whole blood. Those in poor condition, or who had lost more than 20 pounds, had a deficit of about 2,500 to 3,000 c.c. of whole blood. The plasma

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volumes averaged 1,910 c.c. per square meter present surface area in tuberculosis and 1,580 c.c. in the average healthy adults. The increase of plasma volume was not as marked when compared to the usual surface area, 1,760 c.c. to 1,910 cubic centimeters. The average plasma volume (Fig. 2) was 1,780 c.c. per square meter surface area in the patients in good condition, and 1,989 c.c. in the patients in poor condition. Those patients who lost less than 20 pounds in weight had an average plasma volume of 1,611 c.c. per square meter surface area. Those who had lost more than 20 pounds in weight had an average plasma volume of 2,003 c.c. per square meter surface area.

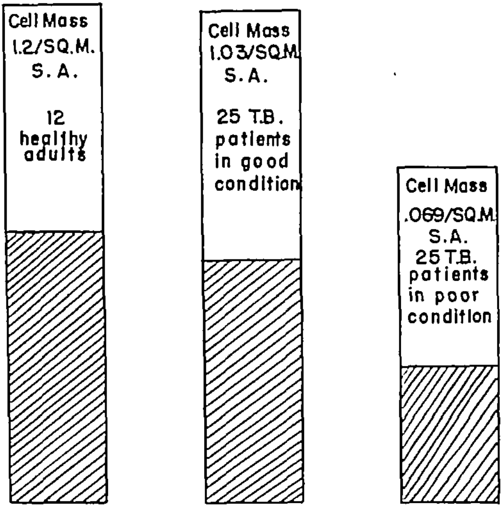

FIG. 1.

The average extracellular fluid (thiocyanate space) volume in the patients with tuberculosis was 11,830 c.c. per square meter of usual surface area and 14,290 c.c. per square meter present surface area. The extracellular fluid averaged 8,150 c.c. in the healthy adults. The extracellular fluid (thiocyanate space) was greater in those patients in poor condition (14,000 c.c. per square meter surface area) than in those in good condition (10,300 c.c.) (Fig. 3). There was an average of 10,900 c.c. extracellular fluid (thiocyanate space) square meter surface area in patients who had lost less than 20 pounds in weight and 13,800 c.c. in those having more than 20 pounds weight loss.

The serum proteins averaged 5.7 Gm. per 100 c.c. in the patients with tuberculosis and 7.1 Gm. per 100 c.c. in the average healthy adults. The average total serum protein, calculated from the plasma volume and serum protein levels, was 168 Gm. in the patients with tuberculosis and 203 Gm. in the healthy adults.

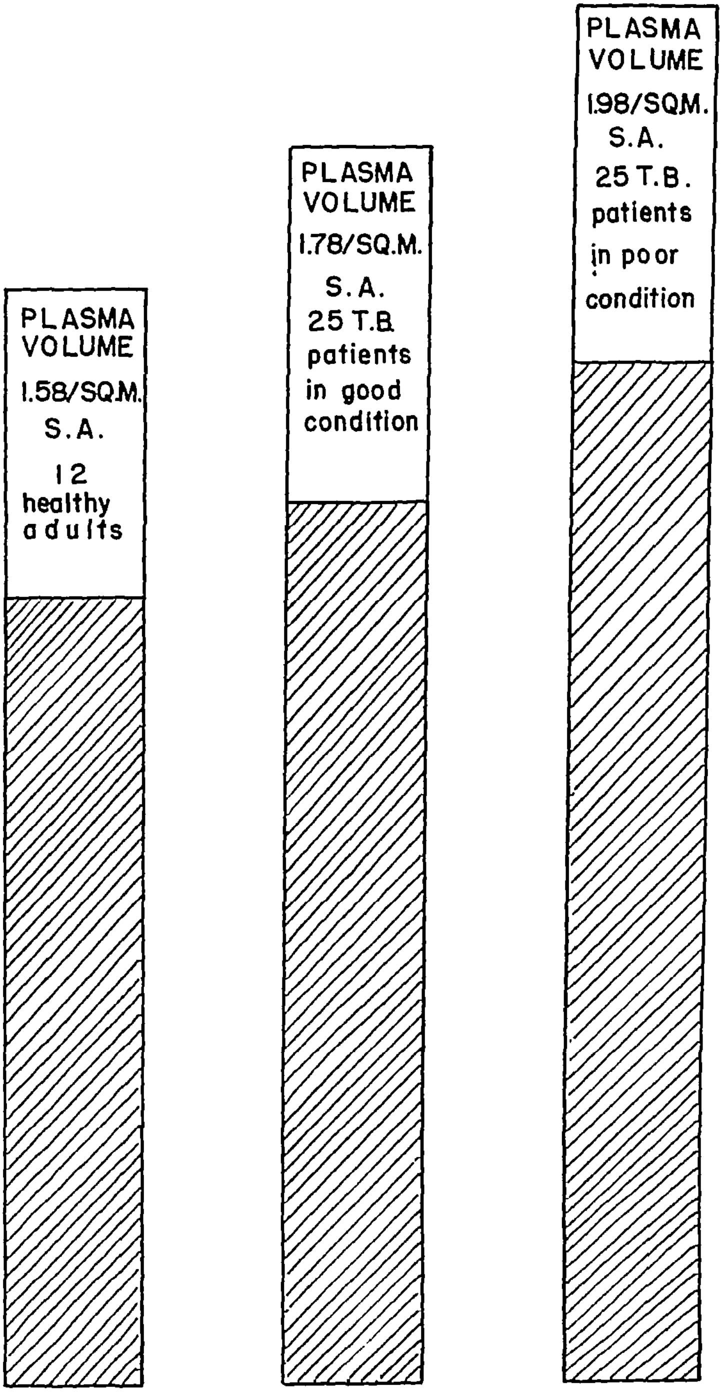
PLASMA VOLUME
1.58/SQ.M. S.A.
12 healthy adults
PLASMA VOLUME
1.78/SQ.M. S.A.
25 T.B. patients in good condition
PLASMA VOLUME
1.98/SQ.M. S.A.
25 T.B. patients in poor condition

Fig. 2.

The average serum albumin for the patients with tuberculosis was 2.9 Gm. per 100 c.c., and for the healthy adults, 4 Gm. per 100 c.c. The total serum albumin, calculated from the plasma volume and serum albumin levels in the patients with tuberculosis averaged 85.5 Gm. and 114 Gm. in the healthy adults. The serum protein levels in the patients in poor condition averaged 5.8 Gm. per 100 c.c., and the serum albumin averaged 2.7 Gm. per 100 c.c. In the patients in good condition these averages were 5.6 Gm. total protein and 2.9 Gm. albumin.

The albumin globulin ratio for the patients with tuberculosis was 1.01 to 1 for those in poor condition, and for those in good condition, 1.04 to 1.

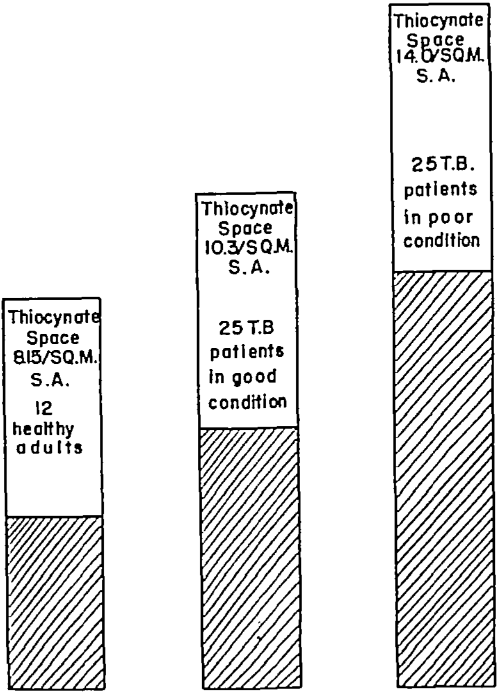

Fig. 1.

DISCUSSION

From these results we can say that there is considerable variation in the extracellular volume, red cell mass, interstitial fluid volume, plasma volume, total protein, and serum albumin in patients with tuberculosis when compared to healthy adults. Some of the laboratory methods used, T-1824 blue dye for plasma volume and thiocyanate for estimation of the extracellular fluid volume, have an estimated error of 10 to 15 per cent, but the variations could not all be accounted for on this basis, as the averages demonstrated definite changes in the patients with tuberculosis.

The results of our investigation suggest that the average patient with tuberculosis has a reduced red cell mass, increased plasma volume, reduced total serum protein, and increased interstitial fluid volume. These changes are more marked in those patients judged to be in poor condition by the criterion used.

The information gathered from this and similar studies[6-15] is important in the treatment of tuberculosis as it serves to re-emphasize the changes in physiology that occur and suggest corrective measures.

There is also a prothrombin deficit in tuberculosis,[16-17] and probably a deficit of other protein compounds, such as antibodies.

The corrective measures suggested from our findings are a high protein intake and transfusions of 2,000 to 3,000 c.c. of whole blood before surgery.

Although these studies were primarily designed to evaluate the preoperative preparation of patients with tuberculosis, the abnormalities found can be utilized in the medical treatment as well.

The increase in plasma volume and extracellular fluid (thiocynate space) volume in these patients is interesting. The greater increase in those patients who have a reduced cell mass suggests a compensatory mechanism.

SUMMARY

1. In patients with tuberculosis studied there was a reduction in red cell mass, an increase in plasma volume, an increase in extracellular fluid (thiocyanate space), and reduced serum total protein and serum albumin, when compared to the healthy adults.

2. These changes were more marked in those patients who were considered to be in poor condition, by the criteria used.

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SUDDEN DEATH OCCURRING IMMEDIATELY AFTER OPERATION IN PATIENTS WITH CARDIAC DISEASE, WITH PARTICULAR REFERENCE TO THE ROLE OF ASPIRATION THROUGH THE ENDOTRACHEAL TUBE AND EXTUBATION

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SUDDEN death is always a catastrophe which is difficult to accept, not only for the patient's family but for the physician as well. When death occurs during an operative procedure it is particularly distressing. Perhaps no calamity, however, is more frustrating than the sudden death of a patient shortly after the completion of an operation at a time when one might assume that the greatest danger was passed. It is our purpose to describe sudden death just after completion of operation in five patients in whom it seemed likely that either aspiration through the endotracheal tube or extubation was an important factor.

CASE REPORTS

Case 1.—This 19-month-old boy had been cyanotic since birth and was markedly incapacitated by his low tolerance for exercise. Examination revealed evidence suggestive of tetralogy of Fallot—moderate cyanosis and clubbing, a harsh systolic basal murmur, a boot-shaped heart by radiologic examination with clear lung fields and diminished vascular markings, right axis deviation on electrocardiography, and marked hemoconcentration. Arterial oxygen saturation was 52 per cent. There was evidence of a right aortic arch. The patient weighed 9.5 kilograms.

Preoperatively the patient was given 32 mg. of pentobarbital, 1 mg. of morphine sulfate, and 0.17 mg. of atropine. Induction was carried out and anesthesia maintained with a cyclopropane-ether-oxygen mixture. During operation 130 mg. of procaine were given slowly by intravenous drip in a 0.1 per cent solution. The operation was begun at 8:45, at which time the pulse was 140 per minute. As soon as the pleural cavity was entered the heart rate slowed to 100, but within a minute after the administration of 0.2 mg. of atropine intravenously the pulse rate rose to 170. Throughout the remainder of the operation the pulse continued at its normal rapid rate and the blood pressure remained constant. The anastomosis between the end of the left subclavian artery and the side of the left main pulmonary artery was begun at 9:55 and was completed at 10:20. Thereafter the color of the patient was improved. By 10:55 the wound had been closed. At this time the level of anesthesia was light. The dressing was applied and good expansion of the lung and the presence of a loud continuous murmur were demonstrated by auscultation. At 11:15 aspiration was carried out through the endotracheal tube and it was withdrawn. Simultaneously the heart sounds ceased, pulse and blood pressure disappeared, and the electrocardiographic tracing which was

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being made demonstrated an instantaneous cessation of electrical activity. The child took two or three coarse gasps and breathed no more. The endotracheal tube was quickly reintroduced and rhythmic expansion of the lungs with oxygen was carried out. The chest wound was reopened and the heart was found in standstill. In spite of prolonged massage and the use of intracardiac adrenalin and Coramine, resuscitation could not be accomplished.

A post-mortem examination was performed. The heart was characteristic of the tetralogy of Fallot. There was present the recently made arterial anastomosis. Otherwise the examination revealed nothing of significance.

Case 2.—The patient was a 6½-year-old child. During an attack of measles, at the age of 2½ years, the child had been examined and the parents had been informed that a cardiac murmur was present. She was subject to frequent upper respiratory infections and was easily fatigued. She weighed 18 kilograms. Upon examination a continuous thrill and murmur were present, most prominent in the second and third interspaces just to the left of the sternum. Blood pressure in the arms ranged between 90 and 108 systolic and 20 and 30 diastolic; blood pressure in the thigh was around 130/40. There was no evidence of decompensation. The electrocardiogram was essentially normal, and roentgenograms were compatible with the diagnosis of patent ductus arteriosus, showing slight left ventricular enlargement and prominent and actively pulsating pulmonary arteries. Even though the findings were rather typical, cardiac catheterization was performed; the pressure and oxygen content studies were also thought compatible with the diagnosis of patent ductus arteriosus.

Preoperatively the patient was given 32 mg. of pentobarbital, 1.7 mg. of morphine sulphate and 0.8 mg. of atropine. There was an easy induction with ether-cyclopropane-oxygen mixture. The operation was begun at 9:45. There was found no true ductus but instead a ligamentum arteriosum which on ligation and section showed a tiny lumen about the size of a silk thread but through which there had been no definite evidence of any flow of blood. A continuous thrill was palpable in the region of the ventricles. The mediastinal pleura was sutured, the lungs re-expanded and the chest wall closed in the usual manner. The left vagus nerve had been blocked with ½ per cent procaine as soon as the chest had been entered. The pulse had ranged between 140 and 160 all through the operation and the systolic blood pressure between 110 and 120. Small amounts of ether and cyclopropane had been used and during the dissection of the ligamentum the patient had received a little 1 per cent sodium pentothal intravenously, a total of 130 mg. having been given. During closure of the chest wall the anesthesia was shifted to a nitrous oxide-oxygen mixture. The operation was completed at 11:05. The dressing was applied and the chest was examined. The lung sounds were normal and the same continuous bruit, which had been heard before operation, was audible. At 11:20 aspiration was carried out through the endotracheal tube and it was removed. Auscultation was being performed at this time and it was noted that the heart sounds ceased as soon as the extubation was accomplished. The tube was immediately replaced. Spontaneous respirations continued for 20 minutes; they were assisted and 100 per cent oxygen was administered. As soon as the disaster occurred the chest was reopened and the heart was found in standstill. Resuscitation was not accomplished in spite of long massage, intracardiac administration of adrenalin and procaine, and electrical stimulation.

A post-mortem examination was not obtained. In view of operative evidence suggesting a ventricular septal defect, it is felt that the erroneous interpretation of the cardiac catheterization studies resulted from the belief, based upon fluoroscopic and radiologic evidence, that the tip of the catheter, at the time one important sample was obtained and pressure studies were made, was in the proximal portion of the pulmonary artery whereas it almost certainly was in the outflow tract of the ventricle.

Case 3.—The patient was an 18-month-old girl who had been cyanotic since birth. She had undergone good development but was markedly limited in her capacity for exercise. She had been seen at the age of 11 months and operation had been deferred until she was older. She had moderate cyanosis, mild clubbing, a basal systolic murmur, a hematocrit of 44, an arterial oxygen saturation of 75 per cent, electrocardiographic evidence of right ventricular preponderance, and typical radiologic findings of tetralogy of Fallot. This diagnosis was further confirmed by angiocardiographic observations.

Preoperatively the patient was given 0.2 mg. of atropine. After Vinethene induction an ether-oxygen mixture was administered. The left vagus was blocked with procaine. The pulmonary artery was large but its pressure was less than 300 mm. of normal saline solution. The operation was begun at 8:00. An anastomosis 3 to 4 mm. in length was made between the aorta and the left main pulmonary artery. Just before the anastomosis was performed 1.5 mg. of curare was given. The pulse rate had remained rather constantly between 130 and 150 and the systolic blood pressure between 100 and 110. Just after the anastomosis had been completed and the clamps on the pulmonary artery released, the heart rate suddenly slowed very markedly. Four-tenths milligram of atropine was given intravenously with transient increase in the heart rate. The rate returned to 140 immediately after administration of an additional 0.4 mg. of atropine and remained at this level during the rest of the operation. A slow drip of 0.2 per cent procaine solution had been started shortly after the pleural cavity was entered. With the second administration of atropine 1.5 c.c. of 2 per cent procaine had also been given. Subsequently another 3 c.c. of procaine was given in divided doses. The vigorous action of the heart and its normal rate continued after the release of the aortic clamp. A continuous thrill was now present. The mediastinal pleura was approximated, the lungs re-expanded, and the chest wall closed. The operation was completed about 9:45. The dressing was applied, the lungs were found to be clear to auscultation and a loud continuous murmur could be heard. Suction was made upon a catheter passed down through the endotracheal tube into the trachea. The patient immediately became pulseless and heart sounds and respirations ceased. The lungs were rhythmically inflated with oxygen and the chest was quickly re-entered. The heart was in standstill. After a little cardiac massage feeble contractions of the heart were initiated. These continued for a short while at a slow rate but normal heart action was not restored in spite of continued massage, intravenous administration of atropine, and intracardiac adrenalin and procaine. A post-mortem examination was not obtained.

CASE 4.—This 3½-year-old boy had the classical findings of coarctation of the aorta. He had enjoyed generally good health, was well developed, and his physical examination was normal except for a loud systolic basal murmur, blood pressure of 130/70 in the arms, unobtainable blood pressure in the thighs, excellent pulses in the upper extremities, and no palpable pulsation in the aorta or the vessels of the lower extremities. Oscillometric values were good in the upper extremities and only a flicker was obtained in the thighs and calves.

One and one-half milligrams of Mu 2006,* a morphine substitute, and 0.25 mg. of atropine were given preoperatively. Ether-oxygen anesthesia was administered after Vinethene induction. The operation proceeded with a great deal more facility than is usually the case in operations for coarctation. The area of coarctation was excised and the ends of the divided aorta were anastomosed with interrupted fine silk sutures. The pulse ranged between 145 and 165 throughout the operation. The systolic blood pressure was rather constantly between 150 and 160 until the aorta was clamped, when it rose to a level of between 180 and 190. When the clamps were removed from the aorta the systolic pressure fell to a level of about 165. The operation was completed in two and one-half hours. The dressing was applied to the wound. The patient was placed on his back and secretions were aspirated from the nose and throat. Aspiration was carried out through the endotracheal tube and a small amount of mucus was obtained. Suction was again applied in the nasopharynx and a second aspiration through the endotracheal tube was productive of only a small amount of mucus. The patient was sufficiently light that he opened his eyes and coughed during this procedure. Just as the endotracheal tube was withdrawn, the patient regurgitated a rather large amount of watery gastric contents which filled his mouth and nose. He was immediately placed in Trendelenburg position and the material was aspirated from the nasopharynx. The patient's color had become ashen gray as soon as the accident occurred and when his color did not improve with a few rapid manual compressions of the bag, suction was applied to a tube placed in the trachea under direct vision and only a slight amount of fluid was obtained. He did not react at all to direct exposure of the larynx or to the aspiration. Only a few feeble gasping respirations occurred. The patient was re-entubated and the lungs rhythmically inflated with oxygen. All of this had only taken a few moments and now it was quite evident that the patient was pulseless and without heartbeat and it seemed likely that cessation of cardiac function had occurred simultaneously with the extubation and regurgitation. The chest was reopened and the heart was found in standstill. Efforts at resuscitation, which included cardiac massage, intracardiac injection of adrenalin and procaine, and electric shock to the heart, were to no avail.

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*Found on clinical study here to be an apparently safe drug.

At post-mortem examination the repaired aorta was found to be in good condition. In the right main-stem bronchus there was a mucous plug which occluded a large portion of the lumen and there was scattered patchy atelectasis of the right lung and to a lesser extent of the left. It was the pathologist's impression that the degree of atelectasis was not sufficient to account for the sudden death and that the opinion of the anesthetist and surgeon that death was due to reflex cardiac arrest was a more likely explanation.

Case 5.—The patient was a 42-year-old woman who had had repeated episodes of upper gastrointestinal hemorrhage since the age of 27. She had had ascites off and on since the age of 28 and had on occasions required paracentesis. She had taken digitalis more or less constantly since the age of 28. Examination revealed auricular fibrillation, ascites, an enlarged firm liver, a very large firm spleen, no increase in venous pressure, circulation time of 14 seconds, normal urinalysis, severe hypochromic anemia, and a bone marrow which was relatively normal. Radiologic examination revealed evidence of an enlarged spleen, esophageal varices, normal pyelograms with intravenously administered Diodrast. Electrocardiographic examination gave evidence of myocardial damage and auricular fibrillation. There was obviously some impairment of liver function. Thymol turbidity was 8.5 units, bilirubin 1 mg. per cent, cephalin flocculation 4+, bromsulfalein 20 per cent retention, prothrombin 42.5 per cent, total proteins 6.42 mg. per cent, albumin 4.22 per cent. After hemoglobin had been raised to a level of 11 Gm. by repeated blood transfusions, she was explored through a combined abdominal thoracic approach.

The preoperative medication consisted of 100 mg. of pentobarbital the preceding night and 10 mg. of morphine sulphate and 0.4 mg. of scopolamine before operation. The induction was accomplished with cyclopropane and oxygen; thereafter anesthesia was maintained with a mixture of ether and oxygen. Splenectomy was very difficult because of the tremendous venous collateral circulation, the large size of the spleen, and the extensive adhesions to it. In spite of great care there was considerable blood loss and 2,000 c.c. of blood were given intravenously. The systolic blood pressure varied between 100 and 110 and continued at this level throughout the long operation until just about the time the spleen was removed. At this time there was a drop to 80/50 but by the time the operation was completed the pressure was 100/60 and had been at this level for over one-half an hour. It had been hoped that a splenorenal anastomosis could be performed. Since, however, the splenectomy itself had proved so extraordinarily difficult and time consuming, it was felt unwise to subject the patient to further operation. After the spleen was removed, it was a tedious matter to secure complete hemostasis and the operation was finally completed 5 hours and 15 minutes after its beginning.

At the time of extubation the patient seemed in excellent condition. The pulse was 112, blood pressure 100/60. As the tube was removed the patient assumed an ashen gray color and pulse and blood pressure were not obtainable. She continued to breathe well and was given oxygen. Intubation was quickly carried out, the chest was opened, the heart massaged and intracardiac injections of procaine, Coramine, and adrenalin were given without success.

TABLE I. DATA CONCERNING CASES OF SUDDEN POSTOPERATIVE DEATH

CASE NUMBER	DIAGNOSIS	INDUCTION	ANESTHESIA	PENTOTHAL	CURARE	PROCAINE	BLOOD LOSS	CARDIAC DIFFICULTY DURING OPERATION	LENGTH OF ANESTHESIA IN HOURS	LENGTH OF OPERATION IN HOURS	PROCEDURE AT TIME OF DEATH
1	Tetralogy	C_3H_6-$(C_2H_5)_2CO_2$	C_3H_6-$(C_2H_5)_2O$-O_2	No	No	Yes	Very little	Early bradycardia responding to atropine	3	2⅙	Extubation
2	Ventricular septal defect	C_3H_6-$(C_2H_5)_2O$-O_2	C_3H_6-$(C_2H_5)_2O$-O_2	Yes	No	No	Very little	None	2	1⅓	Extubation
3	Tetralogy	Vinethene	$(C_2H_5)_2O$-O_2	No	Yes	Yes	Very little	Late severe bradycardia responding to atropine	2¼	1¾	Aspiration
4	Coarctation	Vinethene	$(C_2H_5)_2O$-O_2	No	No	No	Very little	None	3	2¼	Extubation
5	Banti's disease	C_3H_6-O_2	$(C_2H_5)_2O$-O_2	No	No	No	Large	None	6⅓	5¼	Extubation

An arterial transfusion was begun and 1,000 c.c. of blood and 1,000 c.c. of plasma were infused rapidly during the period of cardiac massage. Post-mortem examination was not carried out.

DISCUSSION

There are certain features which are common to all five cases. All were performed under endotracheal anesthesia and in all the to-and-fro absorption technique was used. The actual intubation was easily carried out in each instance. In all the respirations were carefully and regularly assisted by manual compression of the bag. In all the anesthesia was light at the close of the procedure and at the time of the terminal aspiration through the endotracheal tube or extubation. In all the anesthesia was smooth and satisfactory from the standpoint both of surgeon and anesthetist. All of the patients had known cardiac disease. Two had tetralogy of Fallot, one probably a ventricular septal defect and questionably a small patent ductus arteriosus, one a coarctation of the aorta, and one auricular fibrillation and myocardial damage of unknown cause.

Four of the patients were children, one an adult. Induction was carried out with cyclopropane-oxygen in one case, with Vinethene in two, and with cyclopropane-ether-oxygen in two (Table I). Anesthesia was maintained with ether and oxygen in two cases, and cyclopropane, ether, and oxygen in three. In one case a small amount of Pentothal Sodium was administered intravenously, two received dilute procaine solution intravenously, and one of the latter was given curare. Blood loss was considerable in one case, very little in the others. In the exceptional case it was felt that the blood loss was replaced during operation. In three of the patients the exposed vagus nerve was blocked with procaine as soon as the thorax was opened. Two of the patients (Cases 3 and 4) were operated upon in the lateral position. The other three were operated upon lying upon their backs with one side elevated about 30 or 40 degrees.

There had been no evidence of alteration of function of the heart during the operative procedure in three cases, and in a fourth there had been only the briefest relative bradycardia when the chest was first opened and this bradycardia had responded instantly to the use of intravenously administered atropine. The remaining case, however, was one in which there had been an alarming slowing of the heart rate toward the close of the operation. This bradycardia too, however, had responded well to administration of atropine. One of the operations was exceedingly long and difficult and was attended by a transient period of moderate shock though the patient seemed to be in good condition at the completion of the procedure. In the other cases it is felt that the operations proceeded quite smoothly and that the patients stood them very well indeed.

Except possibly for the fact that all had myocardial disease or congenital malformation it would seem that the most plausible factor of significance was the fact that these sudden disastrous changes occurred during suction upon a tube passed into the trachea through the endotracheal tube or during extubation.

Indeed we have met with an apparently similar sudden reflex cardiac death during a period of aspiration through the endotracheal tube shortly after a thoracotomy had been made in the case of a 20-year-old woman who suffered from an infected aneurysm of the ductus arteriosus following its surgical ligation.* In seeking to explain such occurrences, one naturally thinks of vagal reflexes. In operating upon patients with tetralogy of Fallot it is not an infrequent experience that a relative bradycardia occurs sometime during the operation, and that the normal rapid heart rate can commonly be restored by the intravenous administration of atropine. In 1940, Reid and Brace[1] demonstrated electrocardiographic changes in 10 of 35 patients whom they studied during various types of endotracheal manipulation. They attributed these changes to a vago-vagal reflex initiated by the mechanical irritation of the respiratory tract. In his experimental studies with vagal reflexes Sloan[2] found that cardiac arrest could be produced in dogs by vagal stimulation provided rather a marked state of hypoxia existed. Kergin and his associates[3] reported that the use of an oximeter during intrathoracic operations demonstrated that an abrupt drop in oxygen saturation occurred during aspiration through the endotracheal tube. Often levels of from 25 to 30 per cent unsaturation occurred and, unless oxygen were given under pressure, several minutes were required for a return to the pre-existing level of oxygen saturation. My associate and I have confirmed the fall in arterial oxygen saturation during such manipulations. These considerations make it at least a tenable hypothesis that aspiration through and withdrawal of the intratracheal tube at the close of the operation might conceivably bring about cardiac arrest by effecting a transient state of decreased oxygen saturation with resultant increase in susceptibility to vagal reflexes. This mechanism might be brought more easly into play, it would seem, if in addition a state of hypoxia had existed during the operation. In three of the cases reported there was no clinical evidence of oxygen unsaturation during the operation. In the other two, to be sure, cyanosis was present. In both of them, however, as usually happens in cases of tetralogy of Fallot, the color was better during anesthesia than it was beforehand. This was especially true in Case I and there was further noticeable improvement in color once the

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*The patient had been operated upon seven weeks previously, the multiple transfixion and ligation technique of Blalock being used. She was two and one-half months pregnant at the time. Three and a half weeks after operation, there developed hoarseness due to paralysis of the left recurrent laryngeal nerve, chills and fever, and a return of the continuous thrill and murmur and wide pulse pressure which had been present before operation. Large doses of penicillin, aureomycin, and Chloromycetin failed to control the septicemia which was caused by a resistant *Staphylococcus aureus*. After preoperative medication with a small dose of morphine sulfate and 0.8 mg. of atropine, induction was carried out with cyclopropane. Anesthesia was maintained with ether and oxygen. The patient's condition seemed good when the pleural cavity was entered. The mediastinal pleura was incised and a tape placed around the subclavian artery. At this time, electrocardiograms revealed normal sinus rhythm and moderate tachycardia. A few minutes later, the patient was given oxygen under pressure, following which a small catheter was passed into the trachea through the endotracheal tube and suction was applied. It was noted immediately that the heartbeat had disappeared. The pericardium was opened and ventricular fibrillation was observed. Cardiac massage was begun and a moment later a series of four electric shocks was given. The fibrillation continued and a second series of shocks was administered to the heart, followed by the introduction of 40 mg. of procaine into the right ventricle. The fibrillation persisted in spite of the subsequent use of electric shock and intraventricular quinidine. Indeed fibrillation ceased only thirty minutes after its onset as a terminal event and no ventricular contractions could be induced. At post-mortem examination there was evidence of chronic passive congestion of the lungs, liver, and kidneys even though no evidence of heart failure had been observed clinically before operation.

anastomosis was completed. In Case 3 improvement in color with anesthesia and anastomosis was less striking. In none of the cases were oximeter studies carried out during operation and it is recognized that states of arterial oxygen unsaturation can escape clinical detection.

Whether the likelihood of occurrence of such accidents is greater when the anesthesia is light than when it is in a somewhat deeper plane is a matter which deserves careful study. If aspiration of the tracheal contents and extubation are indeed done with somewhat more risk when anesthesia is light such a disadvantage would have to be weighed against such advantages of light anesthesia at the close of operation as the presence of active cough and swallowing reflexes. Though careful aspiration is undoubtedly important it would seem wise to carry out this procedure with as much expedition as possible in order to avoid profound and prolonged decrease in oxygen saturation. One wonders too whether each attempt to aspirate any secretions in the trachea should not be preceded by full inflation of the lungs with 100 per cent oxygen; whether such a practice would reduce the degree of oxygen unsaturation following aspiration could be determined by oximeter studies. One wonders too whether the administration of atropine toward the close of operation before endotracheal manipulation is carried out would confer any additional safety.

It is recognized that the presentation of these cases and this brief consideration of the possible mechanisms involved does not in any sense settle the problem. It is hoped, however, that it may serve to foster a general awareness of such a possible occurrence and to stimulate investigations aimed at throwing more light upon the cause and prevention of these disasters. There is one additional feature which deserves special comment. From reviewing these cases it is evident that completely accurate records of the sequence of events and the time relationships of physiologic changes, therapeutic measures instituted, and any response to such measures will not be preserved unless there exists a prearranged plan according to which one member of the team, and preferably the anesthetist's assistant, assumes among his duties that of recording such data immediately. During periods of such stress time may seem to pass very rapidly and at times very slowly. When one tries with his associates to reconstruct precisely the sequence of events even within a few minutes after death has occurred, one finds it almost impossible to secure agreement as to exactly what happened and when. If a study of such clinical experiences is to be truly helpful, accurate records must be kept. Although it is felt that the essential features of the cases reported are accurate, it is recognized that the accuracy of many details and especially of their time relationships is only approximate.

SUMMARY AND CONCLUSIONS

Five cases of patients with congenital malformation of the heart or of myocardial disease dying suddenly after completion of operation at the time of aspiration through the endotracheal tube or of extubation are presented and discussed, and a sixth case is mentioned in which a similar death occurred shortly after operation was begun. A mechanism which may be responsible is suggested. A plea is made for awareness of this possible occurrence and for clinical and experimental study of its cause and prevention.

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PNEUMOPERITONEUM IN THE DIFFERENTIAL DIAGNOSIS OF DIAPHRAGMATIC HERNIA

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WE HAVE recently employed pneumoperitoneum as a diagnostic aid in four cases of diaphragmatic hernia simulating intrathoracic tumor or cyst. The lesion in these four patients had been detected by routine roentgenography of the chest, and in every instance thoracotomy had been advised as a diagnostic or therapeutic measure.

Increasing use of mass radiological surveys has resulted in the detection of numerous asymptomatic thoracic lesions which present a vexing problem in diagnosis and treatment. Since in many of these lesions thoracotomy is advised primarily for diagnosis, it is apparent that operation could often be avoided if more accurate nonoperative diagnostic measures were available. In the four cases under discussion there was uncertainty not only as to the nature of the lesion but also concerning its position relative to the diaphragm. In these circumstances pneumoperitoneum proved to be a simple and valuable diagnostic maneuver.

The technique of inducing artificial pneumoperitoneum has been described frequently. Our method is essentially that described by Mitchell and associates.[1] After local infiltration anesthesia a one and one-half inch, 19 gauge, short bevel pneumothorax needle with a side opening proximal to its tip is introduced two inches below and to the left of the umbilicus. It is generally easy to detect the entry of the needle into the peritoneal cavity; this is corroborated by gentle introduction of 2 c.c. of air by means of a small syringe. Localized pain at the injection site, or ability to withdraw readily the injected air, suggests that the needle is outside the peritoneal cavity; air injected into the peritoneal space usually passes quickly out of reach of the needle. Injection should not be made if blood is obtained upon aspiration. Neglect of this precaution may lead to injection of air into a solid viscus with possibly fatal air embolism. If the initial air enters freely, a total of 250 to 300 c.c. may be introduced under low pressure; use of a pneumothorax machine is helpful but not essential if care is exercised to avoid forceful introduction when a syringe is used. In two instances we used pneumoperitoneum as an outpatient procedure without interference with the patient's normal activities.

CASE REPORTS

CASE 1.—D. H. (J. H. H. No. 483548). This 14-year-old white schoolboy came to the Johns Hopkins Hospital on Dec. 6, 1948, for advice concerning the management of a "tumor of the left lung" which had been diagnosed elsewhere. He gave a history of

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''asthma'' during the first two years of life followed by ''bronchopneumonia'' which left no obvious ill effects. Except for these early respiratory difficulties he had been considered a strong, healthy, and unusually well-developed boy. Shortly before entry, during a routine roentgenologic survey for tuberculosis, he had been found to have some thoracic abnormality. After full-size roentgenograms had been taken, together with fluoroscopy and barium swallow, his local physician diagnosed a ''tumor of the left lung'' and advised surgical treatment. Before submitting to operation he was brought to the hospital for another opinion. There had been no symptoms of any sort.

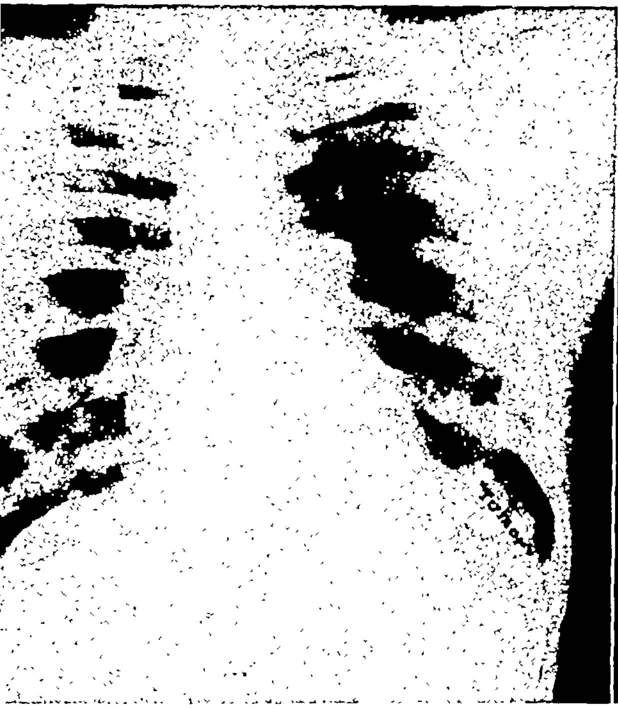

Fig. 1.

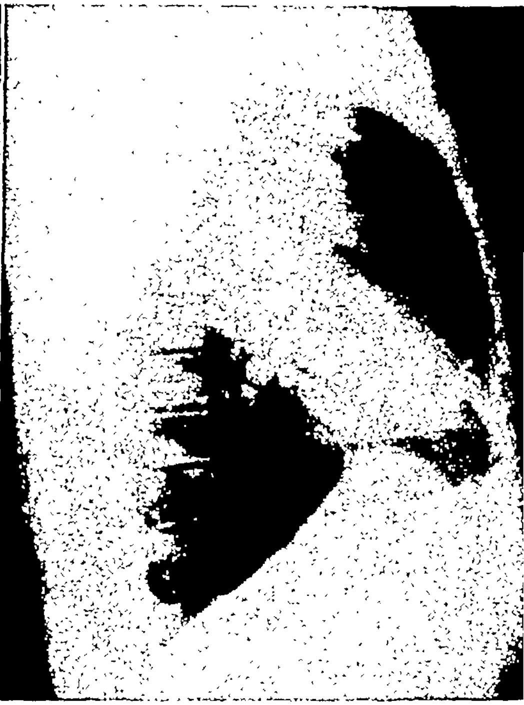

Fig. 2.

Fig. 1.—(D. H.) Roentgenogram in posteroanterior projection. The suspected "tumor" is seen in the region of the left diaphragm.

Fig. 2.—(D. H.) Lateral roentgenogram in extreme inspiration. The anterior portion of the diaphragm fails to descend.

Examination showed a 14-year-old boy nearly six feet tall and weighing 170 pounds. Except for a supernumerary nipple on the left side nothing unusual was observed on physical examination. The heart and lungs appeared normal. Roentgenography of the chest showed a ''density'' at the lower portion of the left lung field between the chest wall and the left lateral border of the heart. In the posteroanterior view this seemed to be sausage-shaped and measured 6.5 by 4 cm. (Fig. 1). A lateral view in extreme inspiration (Fig. 2) showed the lesion to lie anteriorly and apparently below the diaphragm. On fluoroscopy the lateral and posterior portions of the diaphragm seemed to descend well, the anteromedial portion being held up as a rounded knob. This condition is well shown by a left anterior oblique view (Fig. 3) taken in full inspiration. From these studies it seemed unlikely that the lesion lay above the diaphragm, but its exact nature could not be determined.

For this reason a pneumoperitoneum was induced with 250 c.c. of air by the technique previously described. A roentgenogram following this (Fig. 4) demonstrated conclusively that the radiologic appearance which had initially excited concern was due to elevation and

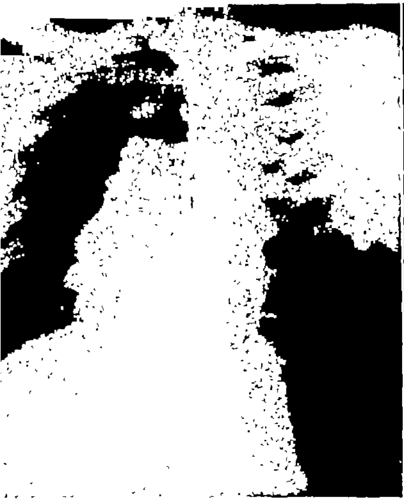

Fig. 3.

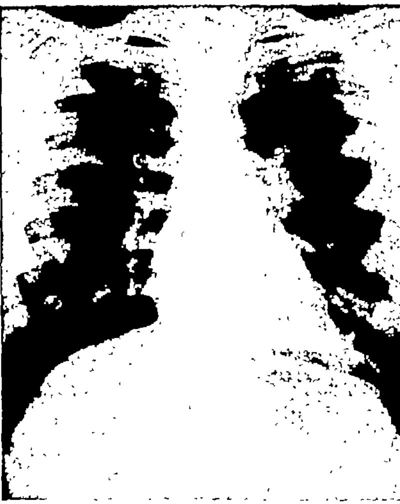

Fig. 4.

Fig. 3.—(D. H.) Left anterior oblique roentgenogram in extreme inspiration. Only the posterolateral portion of the left diaphragm descends.

Fig. 4.—(D. H.) Roentgenogram in posteroanterior projection with pneumoperitoneum. The lateral portion of the left diaphragm is elevated (arrows), but there is no evidence of tumor on the upper or lower diaphragmatic surface.

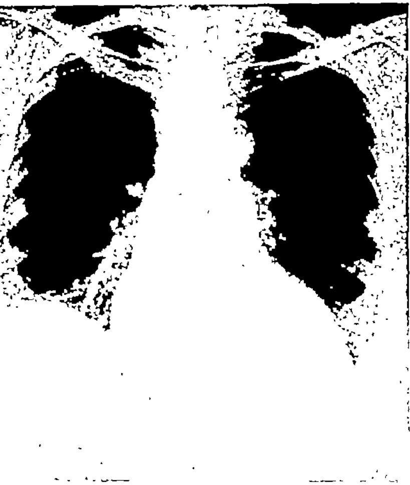

Fig. 5.

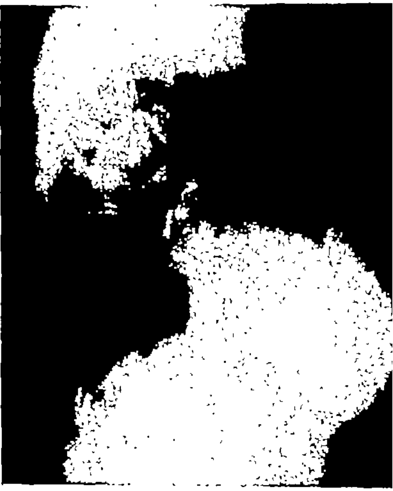

Fig. 6.

Fig. 5.—(X. S.) *Roentgenogram in posteroanterior projection showing smoothly rounded* bulge above the normal contour of the right diaphragm.

Fig. 6.—(X. S.) Lateral roentgenogram showing bulge anterior to midportion of diaphragm overlapping the heart shadow.

paresis of a portion of the left diaphragm, with no evidence of tumor attached to either the upper or the lower surface. The patient's family was reassured, and no treatment was advised. When last seen in October, 1949, he was asymptomatic, and the roentgenologic appearance was unchanged.

Case 2.—X. S. (J. H. H. No. 108672). This 70-year-old housewife was referred to the Private Out-Patient Service on Dec. 21, 1948, after her local physician had detected a roentgenologic abnormality in association with the right diaphragm. For more than twenty-five years she had been followed sporadically in the outpatient department because of menstrual difficulties, pain in the lower abdomen, and a variety of other poorly defined complaints. Attempts at visualization of the gall bladder had failed on four occasions and a cholecystectomy had been advised, but was refused by the patient. In 1941 an upper gastrointestinal series was reported as normal, and no abnormality of the diaphragm was noted.

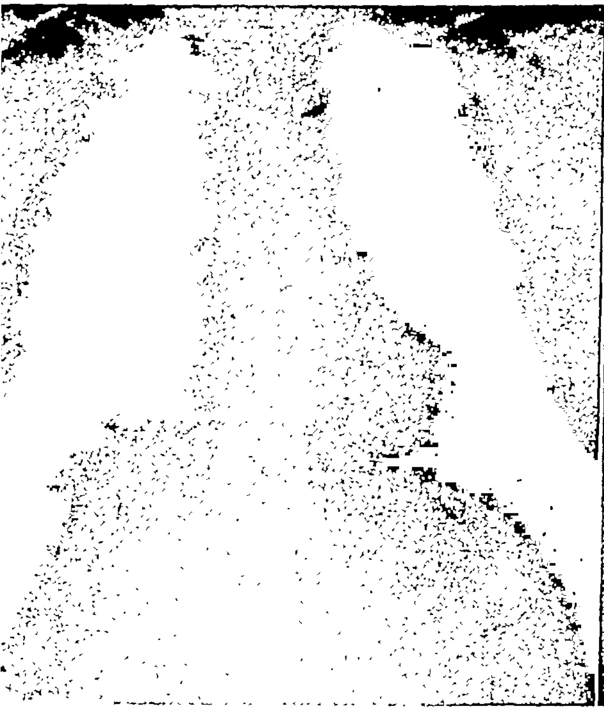

Fig. 7.

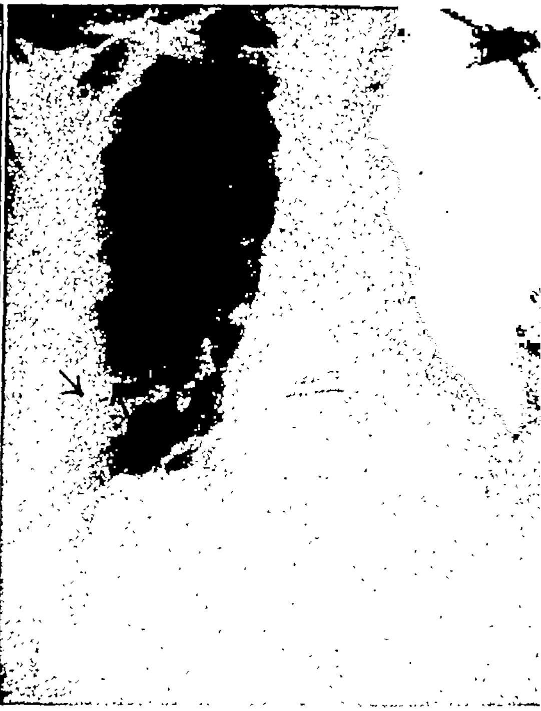

Fig. 8.

Fig. 7.—(X. S.) Roentgenogram in posteroanterior projection during extreme inspiration. The lateral border of the right diaphragm descends well while the central portion remains elevated.

Fig. 8.—(X. S.) Roentgenogram in posteroanterior projection with pneumoperitoneum. The line indicated by arrows represents the thinned-out central portion of right diaphragm with the remaining active portion nearly two interspaces below this.

Physical examination showed only slight hypertension and obesity. There was no abnormality of the chest. Routine laboratory studies of blood and urine were not remarkable.

The patient brought with her a roentgenogram of the chest taken in the posteroanterior projection (Fig. 5). This showed a smoothly rounded bulge above the diaphragm in the right lower chest, apparently separate from the normal contour of the diaphragm. In the lateral view (Fig. 6) this lesion lay anterior to the midportion of the diaphragm, overlapping the heart shadow. On fluoroscopy in extreme inspiration the lateral portion of the diaphragm seemed to descend, but the central portion moved very slightly (Fig. 7).

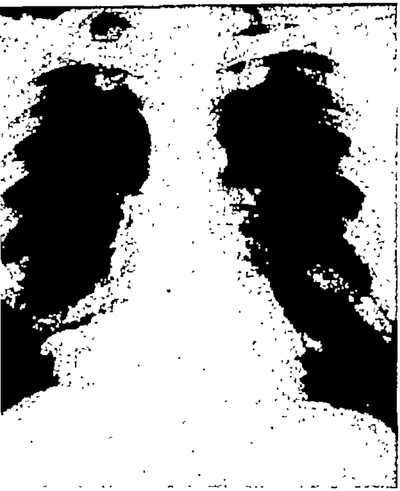

Fig. 9.

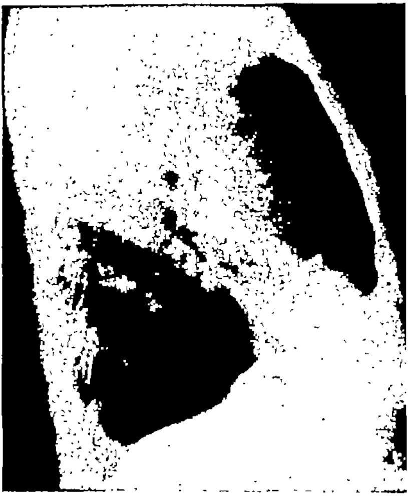

Fig. 10.

Fig. 9.—(I. G.) Roentgenogram in posteroanterior projection showing bulge above right diaphragm continuous with border of the heart.

Fig. 10.—(I. G.) Lateral roentgenogram showing rounded lesion far anteriorly just above normal contour of the diaphragm overlying the heart.

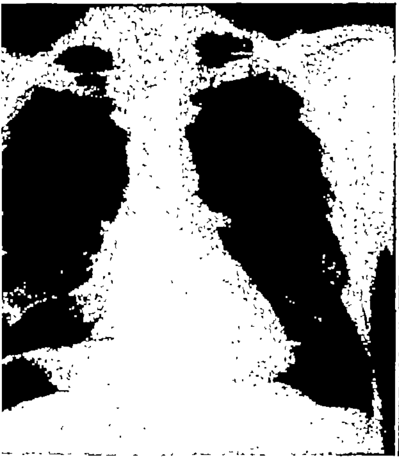

Fig. 11.

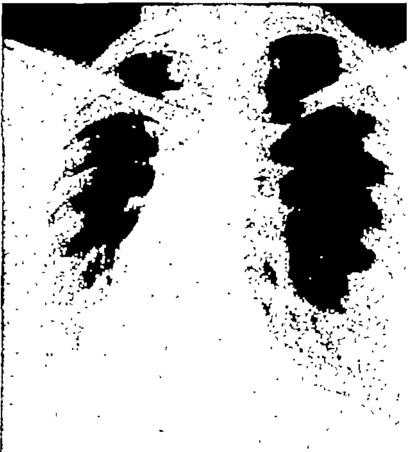

Fig. 12.

Fig. 11.—(I. G.) Roentgenogram in posteroanterior projection with pneumoperitoneum. Both diaphragms are well outlined by air, with the arrows indicating the borders of the hernia through the right diaphragm.

Fig. 12.—(V. G.) Roentgenogram in posteroanterior projection showing shift of heart and mediastinum to right. The bulging of the mass in the lower right chest obliterates the normal cardiophrenic angle

Pneumoperitoneum with 250 c.c. of air was induced in the outpatient department. A roentgenogram (Fig. 8) demonstrated air outlining the highest limits of the previously noted density. This upper limit was bounded by a thin line presumably representing a thinned-out portion of diaphragm with the remaining active portion lying two interspaces below this. This 70-year-old patient suffered no ill effects from the procedure. She was reassured and no treatment was recommended.

CASE 3.—I. G. (J. H. H. No. 493822). This 48-year-old white pharmacist was admitted to the Johns Hopkins Hospital as a patient of Dr. George Finney on Mar. 15, 1949. Three months before admission a ''mass in the right lung field'' had been discovered by his local physician following routine roentgenography of the chest. He gave a history of bronchopneumonia at the age of 8 years. There had been a nonproductive cough for many years. Except for this there had been no cardiorespiratory symptoms. Bronchoscopy and bronchography elsewhere had given no significant information concerning the nature of the ''mass'' in the right lung area.

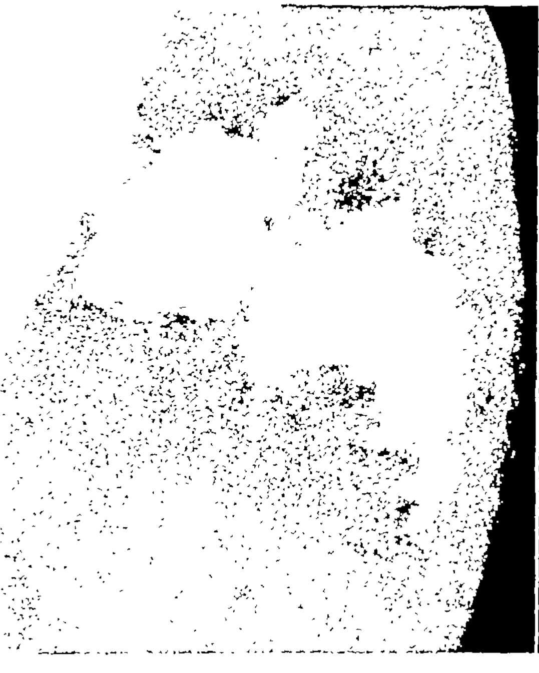

Fig. 13.

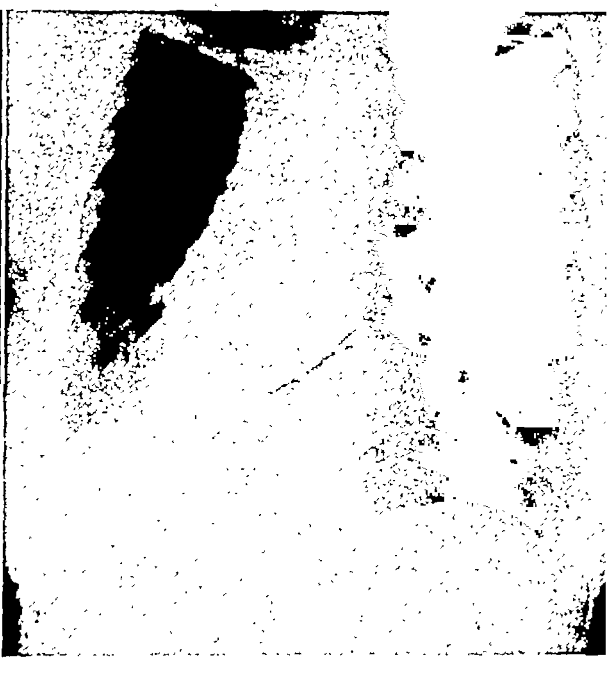

Fig. 14.

Fig. 13.—(V. G.) Lateral roentgenogram showing rounded retrocardiac density low in the chest. Before admission this had been diagnosed as a posterior mediastinal tumor or cyst.

Fig. 14.—(V. G.) Roentgenogram in posteroanterior projection, with pneumoperitoneum. Air is visible under the left diaphragm and at the lateral border of the density in the right cardiophrenic angle.

On admission he was found to be an adequately nourished man who did not seem ill. There were multiple telangiectatic lesions of the skin. A few respiratory râles and rhonchi were detected at the base of the right lung. Temperature, pulse, respirations, and routine laboratory studies of blood and urine showed no abnormality.

A roentgenogram of the chest in the posteroanterior projection showed the lesion which had initiated the investigation (Fig. 9). It appeared to be above the diaphragm, close to the right border of the heart. A lateral view showed it to lie anteriorly (Fig. 10) just behind the sternum.

A gastrointestinal series and barium enema showed no evidence of gastrointestinal contents in the region of the lesion. There were a few diverticula of the ascending colon.

Diagnostic pneumoperitoneum was performed, 300 c.c. of air being introduced by the usual technique. A roentgenogram following this showed both diaphragms well outlined by underlying pockets of air which also filled the region of the "mass" near the right border of the heart (Fig. 11). Since barium studies had shown no stomach or bowel in the hernia, and because he was essentially asymptomatic, no treatment was advised.

CASE 4.—V. G. (J.H.H. No. 535604). This 49-year-old white woman entered the Johns Hopkins Hospital on April 24, 1950, because of the discovery of a mass in her right chest thought to be a posterior mediastinal tumor or cyst. She had always enjoyed good health, with no gastrointestinal or cardiorespiratory symptoms.

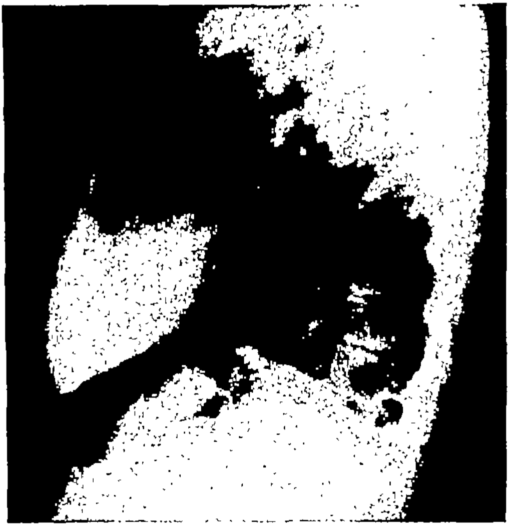

Fig. 15.—(V.G.) Lateral roentgenogram with pneumoperitoneum. There is no air at the apex of the rounded density for reasons detailed in the text.

Two months before entry, after a severe respiratory illness, the patient began to have pain in her right shoulder and scapula and a cough productive of small amounts of mucus. Her physician detected dullness at the base of the right lung and a friction rub, and treated her with penicillin. Roentgenologic study of the chest revealed a retrocardiac mass on the right, and a mediastinal shift to the right. The cough disappeared in a few days, and she was left with only a pain in the right shoulder. This pain subsequently shifted to the left shoulder; hence, it seems unlikely that it was related to the disease process in the region of the right diaphragm. A review by her physician of chest roentgenograms made in 1946 and 1949, when the patient was perfectly well, revealed the same right-sided retrocardiac mass and a shift of the mediastinum to the right. The discovery of a "chest tumor" in this patient was therefore an accidental occurrence during the investigation of a respiratory illness.

On admission to the hospital the only significant physical signs were dullness to percussion and diminution of breath sounds and tactile fremitus over the posterior right chest below the angle of the scapula. The dullness did not shift on deep inspiration. The trachea was deviated to the right.

On April 29, 1950, the right chest was entered through the bed of the eighth rib. All lobes of the lung were found expanded which ruled out atelectasis as a cause of the mediastinal shift. The lower lobe was everywhere adherent to the diaphragm by filmy adhesions. When the adhesions were freed the mass was seen to be a knob of liver covered by a thin sac. The failure of the pneumoperitoneum to outline the mass completely was due to inclusion in the sac of much of the bare area of the liver, which is, of course, extraperitoneal (Fig. 16).

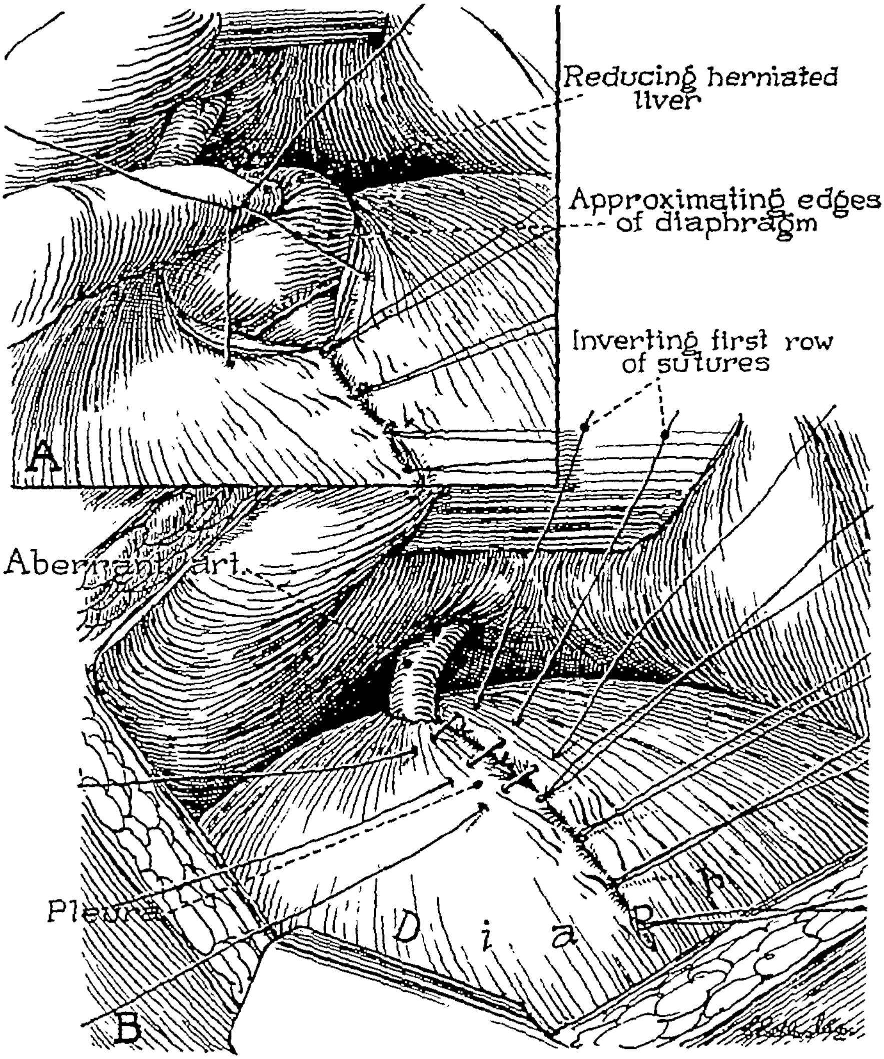
Reducing herniated liver
Approximating edges of diaphragm
Inverting first row of sutures
A
Aberrant art.
Pleura
D i a p h
B

Fig. 18.—*A*., Reduction of the hernia is being completed as suturing of defect in diaphragm progresses. *B*., Second row of sutures is nearly complete. Residual defect for passage of aberrant artery is shown.

The sac was thin and transparent, and seemed grossly to contain no muscle fibers. It appeared to represent diaphragmatic pleura fused with peritoneum except at the bare area of the liver where the peritoneum is deficient. Anteriorly, laterally, and medially the hernial ring was bounded by thick diaphragmatic muscle. The defect was horseshoe shaped and lay in the posterolateral portion of the diaphragm. Passing through the posterolateral part of the hernial opening was an artery about 4 mm. in diameter, which arose below the diaphragm and arborized over the inferior surface of the lower lobe of the right lung (Fig 17, *A*).* When the hernial ring was dissected free, the projecting liver was seen to be a boss with a broad pedicle constricted by the diaphragmatic ring (Fig. 17,*B*). After this projection was pushed down below the diaphragm, a two-layer closure of the diaphragmatic defect was carried out with interrupted sutures of silk. The line of suture began at the central tendon and progressed toward the posterolateral chest wall, approximating the limbs of the horseshoe until a triangular defect 2 cm. in size remained (Fig. 18). Further closure would have produced undesirable tension on the suture line and would also have necessitated division of the anomalous artery. For these reasons the small posterolateral defect was left, and the chest was closed with intercostal catheter drainage. The phrenic nerve was not crushed.

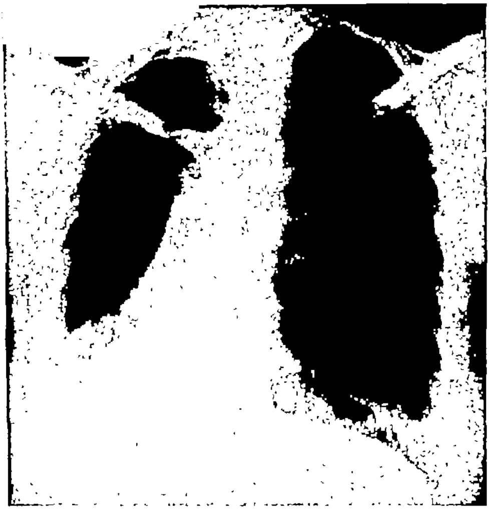

Fig. 19.—(V. G.) Roentgenogram in posteroanterior projection made six weeks after operation. There is some elevation of the right diaphragm, and the mediastinum is still shifted to the right. The previously herniated liver is no longer seen.

The patient was ambulatory from her sixth postoperative day. Fluoroscopy on the thirteenth postoperative day showed no change in the mediastinal shift to the right. The right lung base was hazy, but there was no evidence of a hernia. The patient left the hospital on the fourteenth postoperative day. A film made six weeks after operation (Fig. 19) shows the diaphragm intact and somewhat elevated, and the mediastinum still shifted to the right.

It should be noted that this patient had a right cervical rib, a congenital diaphragmatic hernia, and an anomalous artery to the lower lobe of the right lung, a remarkable triad of developmental abnormalities.

DISCUSSION

The usefulness of any diagnostic test is determined by its applicability and by the degree of attendant inconvenience or danger when measured against the value of the information obtained. By these criteria there seems to be a place for pneumoperitoneum in the investigation of certain lesions.

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*Such anomalous arteries to the lung have been discussed by Bruwer, Clagett, and McDonald.[5]

In regard to the applicability of diagnostic pneumoperitoneum, there have been many suggestions for its use. It has been considered applicable in outlining the pelvic organs,[3] in locating subphrenic abscess,[4] in distinguishing hepatic abscess from nodular cirrhosis,[5] and in determining whether inflammatory lesions lie above or below the diaphragm.[3] It seems ideally suited for the determination of the position of any lesion relative to the diaphragm, and there is occasional reference to its use in the voluminous literature on so-called eventration of the diaphragm. Most publications on the subject of eventration are confusing or misleading, and for an extended critical discussion of the topic one should consult the reviews of Korns[6] and Reed and Borden.[7]

Leaving etymology and historical perspective to such reviews, one may, perhaps arbitrarily, consider eventration as a *uniform* elevation of one leaf of the diaphragm. Such elevation may be congenital or may occur as a result of various poorly understood lesions of the phrenic nerve or the diaphragm itself. Elevation of the hemidiaphragm following surgical crushing of the phrenic nerve is not considered an eventration, although it fulfills the usual criteria for this entity. In contrast, any *localized* protrusion through the diaphragm, whether it be congenital or acquired, and irrespective of the presence or absence of a sac, may properly be called a diaphragmatic hernia. Straddling the issue by the use of such a term as "localized eventration" for what is essentially a hernia seems to us a further source of confusion between two clearly delimited entities. The distinction between eventration and hernia of the diaphragm by roentgenographic study after pneumoperitoneum was suggested by Schlect and Wels,[8, 9] who actually employed the procedure. By means of pneumoperitoneum Walton[10] demonstrated an eventration, and Pacheco[11] proved that a mass in the lower part of a patient's right chest was situated below the diaphragm. He believed this to be an hydatid cyst, and only at thoracotomy was it shown to be a diaphragmatic hernia containing liver and gall bladder. A number of other authors[12, 13, 14] suggested that such a maneuver might be used, but made no mention of actual clinical application.

It is only recently that the radiological "survey lesion" in the lung fields has rendered diagnosis and therapy in this area much more complex. Most of these lesions are clearly above the diaphragm, but those low in the lung fields can be confused with subdiaphragmatic processes. Clark and co-workers[15] mentioned the use of pneumoperitoneum in distinguishing such pulmonary lesions from diaphragmatic hernia. Lilienthal[16] and Wolfson and Goldman[17] have reported operations for supposed mediastinal tumor in which diaphragmatic hernia containing liver was found. In each case the authors noted that a pneumoperitoneum would have led to the proper diagnosis before operation. Patients reported by Harrington,[18] Gross,[19] Wagner,[20] and Bradley and Greiner[21] were thought to have a mediastinal cyst or tumor, and at operation or at autopsy all were found to have diaphragmatic hernia. In these cases it seems likely that roentgenography after pneumoperitoneum would have led to the proper diagnosis without operation.

After pneumoperitoneum was introduced into this country as a diagnostic aid by Stein and Stewart,[22] it enjoyed a brief popularity which was diminished by Case's report[23] of four deaths gathered from the literature and from extensive correspondence with physicians. Eight additional deaths are noted up to 1947 in the report of Mitchell and his associates[1] on therapeutic pneumoperitoneum, and there are doubtless a number of unreported fatalities. In 1919 Schlecht and Wels[9] reported a death due to perforation of a gangrenous area of the stomach three weeks after pneumoperitoneum. This appears to have been due to strangulation of the stomach in a diaphragmatic hernia, and the authors considered it unrelated to the pneumoperitoneum.

We agree that the procedure is not without danger; a fatal instance of air embolism occurred some years ago in this hospital during pneumoperitoneum designed to demonstrate a subphrenic abscess. In this case the technical precautions noted previously were not observed. Most of the reported deaths are due to air embolism which can be readily avoided. Despite the reported deaths the procedure appears fairly safe when judged in relation to the thousands of patients in whom therapeutic pneumoperitoneum is now being used repeatedly.

SUMMARY

Pneumoperitoneum and subsequent roentgenography have been employed in four patients who were thought to have a low-lying intrathoracic tumor or cyst. All were shown to have diaphragmatic hernia.

A carefully performed diagnostic pneumoperitoneum is a relatively safe procedure which may enable one to avoid the hazard and inconvenience of diagnostic thoracotomy.

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CARTILAGINOUS TUMORS OF RIBS AND STERNUM

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CARTILAGINOUS tumors of the bony chest wall are only infrequently seen but are the most commonly encountered type of primary tumor in this region. Any tumor that occurs in bone may also arise in the ribs and in the sternum. Because of the singular problems that cartilaginous tumors of the chest wall present regarding pathology, prognosis, and surgical treatment, they should be given special study.

Reviews of tumors of the bony thorax are legion. In 1897, Paget[45] reviewed thirty-three tumors of the ribs and sternum, most of which were cartilaginous. Hedblom,[27, 28] in 1921 and in 1933, collected a large number of lesions of ribs and sternum, but lack of adequate follow-up as well as the inclusion of metastatic tumors and non-neoplastic conditions decreased the value of his work. Heuer[29] and more recently Kinsella, White, and Koučky[30] have presented reviews of a variety of sternal lesions; and in 1939 Harper[21] studied benign cartilaginous tumors of the rib. In 1942, Sommer and Major[63] collected 66 cases of primary neoplasms of the bony thorax which had appeared in the literature after Hedblom's review, and added fifteen cases of their own. Of this series of eighty-one cases, thirty-one (38.4 per cent) appear to be of cartilaginous type, both benign and malignant. Dorner and Marcy[11] in 1948 presented a similar review of the intervening years totaling twenty-three cases. Fifteen (65.2 per cent) of these were of cartilaginous origin and were designated as chondroma, chondrosarcoma, osteochondroma, and osteochondrosarcoma. Most of the remaining tumors in these series were giant cell tumor, Ewing's sarcoma, osteoma, hemangioma, fibrosarcoma, and myeloma.

We have not encountered a case of osteogenic sarcoma of rib or sternum at Barnes Hospital, and we feel that reported cases which are free of the suspicion of being chondrosarcomas are extremely rare. This low incidence of osteogenic sarcoma of rib and sternum is remarkable in view of the preponderance of osteogenic sarcoma over chondrosarcoma in other sites. Phemister,[52] in reviewing sixty-one bone sarcomas of all sites, found ten (16.4 per cent) were chondrosarcomas.

Primary malignant tumors of rib and sternum comprise about 10 per cent of all primary malignant bone tumors.[43] In Geschickter's large series of 488 cartilaginous tumors,[17] location in ribs and sternum was found in only nineteen (3.8 per cent). Of those tumors classified as benign (osteochondroma, chondroma), 3 per cent were in the chest wall; and of those classed as malignant (primary and secondary chondromyxosarcoma), 5.8 per cent were in the bony thorax.

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From the pathologic aspect there has been a great tendency to "underdiagnose" these tumors. Many chondrosarcomas, both in our series and in the reported cases, were originally called benign. Lichtenstein and Jaffe[37] have refined the histologic diagnosis of cartilaginous tumors by establishing criteria of malignancy which must be found in viable actively growing portions of the tumor: (1) many cells with plump nuclei, (2) more than an occasional cell with two such nuclei, and (3) giant cartilage cells with large single or double nuclei or with clumps of chromatin.

Regarding the pathogenesis of these tumors, Ewing[12] and others before him back to Virchow considered that cartilaginous tumors could arise from normal cartilage or from cartilaginous rests displaced during enchondral osteogenesis. Jaffe and Lichtenstein[33] agreed that displaced cartilaginous rests, usually found in the metaphyses, are possible sites of origin of enchondromas; however, Jaffe[32] hypothecated that exostoses (osteochondromas, ecchondromas) are the result of disordered periosteal activity, and more specifically, that the periosteum may form cartilage instead of bone. This hypothesis would explain the occasional origin of cartilaginous tumors in rib distant from the metaphysis.

Table I shows the eighty-five cases of chondroma, osteochondroma, and chondrosarcoma taken from the literature with the addition of our eleven cases. This does not presume to be a complete review of the literature, but only an attempt to collect enough cases so that some statistical conclusions might be drawn. The low incidence of these types of tumor is indicated by the numerous authors reporting a single case. The 85 cases that are reviewed were reported in 49 articles.

Group I (60 cases) consists of tumors diagnosed as malignant, and in Group II (36 cases) are those that were called benign. Our cases are all in Group I. Certain difficulties, in addition to the tendency of many authors to underdiagnose the tumors, were encountered in this review. Before the Bone Sarcoma Registry of the American College of Surgeons[12] accepted the diagnosis of chondrosarcoma as a separate entity from other primary malignant tumors of bone, there was considerable confusion in diagnostic terminology. There was also failure in many cases to appreciate the distinction between enchondromatous and ecchondromatous neoplasms.

Undoubtedly many of the tumors in Group II are malignant. Suspicion is cast particularly on those cases that recurred, often several times (Cases 69, 77, 83, 88, 89, 94). Auchincloss' case (Case 73) must be suspected of being a malignant tumor because of the "identical bony nodule" in the lung. In other instances (Cases 62, 72, 96), the authors themselves imply that there was a possibility of malignancy, even though the diagnosis of benign tumor was made. Because of these cases, we are skeptical about the entire group, particularly the many with poor follow-up. The most convincingly benign tumor is that reported by Simpson[61] (Case 87). This tumor was heavily ossified and remained radiographically unchanged over a period of seventeen years. Graham[19] believes that most of the cartilaginous tumors of the chest wall are malignant.

CLINICAL FEATURES

Age.—The mean age of the patients at the onset of symptoms in the malignant Group I was 41.4 years with a range from 11 to 74 years (Fig. 1). In Group II the mean age at onset was 32.6 years with a range from birth to 60 years (Fig. 2).

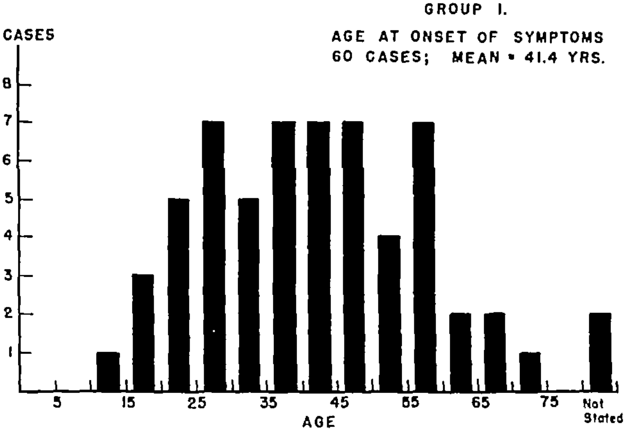

FIG. 1.—Age incidence: chondrosarcoma of rib and sternum.

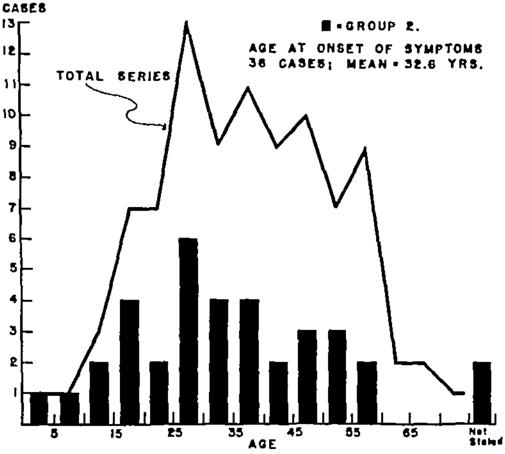

FIG. 2.—Age incidence: so-called benign tumors of rib and sternum and of the total group.

SUMMARY OF CASES

Group I: Chondrosarcoma

AUTHOR	YEAR	SEX	AGE ONSET (YR.)	INITIAL SYMPTOMS	LOCATION	DURATION UNTIL FIRST TREATMENT	OPERATION	CATEGORY OF OPERATION	OUTCOME	AUTHOR'S DIAGNOSIS
1. Hedblom[27]	1921	M	28	Tumor	3, 4, and 5 ribs, left, anterior	15 yr.	Partial excision	2	Died 13 mo. of recurrence	Osteochondrosarcoma
2. Hedblom[27]	1921	M	40	Tumor	3, 4, and 5 ribs, anterior	6 yr.	a. Removal b. Resected ribs 3, 4, and 5 and stripped off pleura without tearing it. c. Removal d. Removal e. Two other operations not detailed	1 4 1 1 1 1	a. Recurrence 3 mo. b. Recurrence c. Recurrence d. Recurence e. Recurrence f. Recurrence Died of recurrence 6 yr. after initial operation	Myxochondrosarcoma
3. Harrington[24]	1927	M	58	Tumor	4th rib, right	6 mo.	Resection with ⅓ of 4th rib	4	Living and well 8 months	Chondrosarcoma grade I
4. Harrington[24]	1927	M	54	Pain	8th rib, right anterior axillary line	2 mo.	Resection with 8th and 9th ribs; pleura not taken	4	Living and well 1 yr. 8 mo.	Chondrosarcoma
5. Lockwood[38]	1928	F	50	Tumor	1st and 2nd ribs, right, anterior	8 mo.	a. Tumor removed down to rib; rib curetted b. Several recurrent nodules found and curetted c. Pleura and lung involved; tumor curetted	3 3 3	a. Recurrence 1½ yr. b. Recurrence 2 yrs. 10 mo. c. Recurrence 8 mo. Living with recurrence 6 yr. after initial operation	Chondrosarcoma
6. Denk[10]	1930	F	44	Pain Horner's	2, 3, 4, ribs posterior, right	1 yr.	Paravertebral resection of ribs 2, 3, 4, and scraping tumor off vertebrae	3	Died pulmonary embolism 6 days postoperatively	Chondrosarcoma
7. Phemister[52]	1930	M	18	Tumor	2, 3, 4, 5, 6, 7, right anterior	2 yr.	Only large external mass removed	2	Died 9 mo. metastates lung	Chondrosarcoma

8. Zinninger[69]	1930	M	20 plus	Tumor	7th rib, right	Several yr.	Resected 3 ribs with considerable pleura	5	Died postoperatively	Chondrosarcoma
9. Geschickter[17]	1931	M	47	Tumor	3rd rib, anterior	7 yr.	Resected 3 times; details not given	1 1 1	Recurrence Recurrence Recurrence Died 6 yr. after initial operation	Secondary chondromyxosarcoma
10. Geschickter[17]	1931	F	30	Tumor and pain	Ribs, right	1 yr.	Resection; no details given	1	No follow-up	Secondary chondromyxosarcoma
11. Geschickter[17]	1931	F	17	Pain and tumor	9th rib	4 yr.	Resection 2 times; no details given	1 1	Recurrence Living and well 9 yr. after second operation	Secondary chondromyxosarcoma
12. Geschickter[17]	1931	F	33	Tumor	Ribs at sternum	1 yr.	Curettement 3 times	3 3 3	Recurrence Recurrence Recurrence Lost to follow-up	Secondary chondromyxosarcoma
13. Geschickter[17]	1931	M	40	Tumor	Upper sternum	3 yr.	Partial excision	2	Living and well 9 yr.	Secondary chondromyxosarcoma
14. Geschickter[17]	1931	M	42	Multiple tumors	Ribs, multiple	14 yr.	Excision, no details given	1	Dead, 10 mo.	Secondary chondromyxosarcoma
15. Geschickter[17]	1931	M	50	Rib fracture	Ribs	—	Excision; no details given	1	Dead; 5 weeks; local recurrence	Secondary chondromyxosarcoma
16. Vinnay[67]	1931	M	25	Tumor	8, 9, 10th ribs, lateral	4 yr.	Removal with 8-10th ribs	5	Living and well 3 yr.	Chondrosarcoma
17. Heuer[29]	1932	F	20	Pain	Sternum	16 yr.	a. Removal b. Removal with pleura and lower two-thirds of sternum; tumor ruptured during removal	1 3	a. Recurrence 9 yr. b. Recurrence 1 yr.	Chondromyxoma, benign
							c. Exploration; unresectable	2	c. Metastasis to lung; local recurrence. Living with recurrence 12 yr. after initial operation	

TABLE I—CONT'D

AUTHOR	YEAR	SEX	AGE ONSET (YR.)	INITIAL SYMPTOMS	LOCATION	DURATION UNTIL FIRST TREATMENT	OPERATION	CATEGORY OF OPERATION	OUTCOME	AUTHOR'S DIAGNOSIS
18. Cabot[6] Case 10202	1933	M	39	Tumor	6th to 10th ribs right anterior	3 yr.	Exploratory thoracotomy; *tumor not resectable*	2	Died from operation	Osteogenic sarcoma (chondrosarcoma)
19. Matson[39]	1934	F	26	Pain	7th rib, left, posterior (at angle)	11 mo.	Resection of tumor with adjacent ribs and lung attachment	5	Living and well 1 yr. 3 mo.	Osteogenic sarcoma
20. Papin[40]	1934	--	43	Pain	3, 4, 5 ribs, anterior	--	Removal of 3-5th ribs	5	Living and well 14 mo.	Chondromyxosarcoma
21. Campbell[8]	1935	M	36	Tumor	Anterior ribs	2 yr.	Partial excision	2	Dead 3 yr. metastases	Malignant chondroma
22. Halpert & Davis[20]	1935	M	40	--	12th rib, right	--	Removal from rib	1	Dead 1 yr. recurrence and metastases	Chondrosarcoma
23. Roberg[57]	1935	F	37	Pain	Middle of sternum	13 mo.	a. Resection of midsternum after bilateral division of 2-5th cartilages b. Resection of manubrium with portions of right clavicle and portions of 1st and 2nd ribs	4 4	a. Recurrence at 3 mo. in manubrium b. Recurrence at 11 mo. in scar and pulmonary metastases	Chondrosarcoma
24. Roberg[57]	1935	F	45	Pain	Left chest	5 yr.	a. Tumor and piece of rib removed b. Re-excision	1 1	a. Recurred 2 weeks b. Living but moribund with orange-sized recurrence at 6 weeks	Chondromyxosarcoma

25. Robert-[58] Didier	1935	F	29	Tumor	Lower sternum	2 yr.	a. Resection	4	a. Recurrence 2 mo.	Chondro-osteo-sarcoma
							b. Resection of recurrence	4	b. Recurrence 5 mo.	
							c. Resection of recurrence	4	c. Living and well 3 mo. after third operation	
26. Speed[65]	1936	M	50	Tumor	2, 3, 4 ribs, left anterior	3 yr.	Removal in 2 pieces	3	Living 6 mo. with recurrence	Chondrosarcoma
27. Speed[65]	1936	M	35	Tumor	3rd rib, right anterior	2 yr.	a. Removal	1	a. Local recurrence 1 yr.	Chondromyxo-sarcoma
							b. Block dissection of chest wall; intrathoracic portion scooped out	2	b. Recurrence in scar at 3 yr. Dead 6 yr. metas. scalp and thigh	
28. Cabot[7, 9] Case 24212	1938	M	22½	Tumor	Left chest, posterior	3 mo.	a. Excision of tumor and part of rib, left	4	a. Recurrence at 13 mo.	Chondrosarcoma
							b. Excision of rib stump and adjacent rib	4	b. Recurrence at 8 mo.	
							c. Excision of tumor in scar	3	c. Recurrence at 27 mo.	
							d. Excision of entire remaining rib	4	d. No local recurrence Met. to RLL	
							e. Right lower lobectomy with excision of portion of diaphragm and chest wall	5	e. Recurrence at 4 mo.	
							f. Excision of right chest wall and ribs 8-12 en bloc with leaf of diaphragm	5	f. Died of recurrence at 6 mo. (71 mo. after onset)	
29. Janes[34]	1939	M	63	Tumor	Right 3rd costochondral junction	1 mo.	Removed whole growth	4	Living with recurrence at 1 yr.	Chondrosarcoma
30. Bradshaw[5] & Chodoff	1940	F	45	Tumor	Upper sternum	4 yr.	Upper sternum and inner ends of both clavicles removed	4	Living and well 23 mo.	Chondrosarcoma

TABLE I—CONT'D

AUTHOR	YEAR	SEX	AGE ONSET (YR.)	INITIAL SYMPTOMS	LOCATION	DURATION UNTIL FIRST TREATMENT	OPERATION	CATEGORY OF OPERATION	OUTCOME	AUTHOR'S DIAGNOSIS
31. Janes[35]	1941	M	30	Tumor	9 and 10th ribs, right anterior	15 yr.	Excision under local anesthesia	3	No follow-up	Chondrosarcoma
32. Janes[35]	1941	M	58	Tumor	9, 10, 11th ribs, right anterior	1½ yr.	Removal of tumor with adjacent abdominal and chest wall. Tumor stripped from pleura and peritoneum	4	Living and well 18 mo.	Chondrosarcoma
33. Janes[35]	1941	M	37	Tumor	4th rib, posterior left	2 yr.	Removal of tumor, pleura, posterior portion of 1st to 7th ribs	5	No follow-up	Chondrosarcoma
34. Janes[35]	1941	M	16	Cough	9th rib, posterior left	--	Biopsy only	0	Dead 1 mo. after onset	Chondrosarcoma
35. Mayer[41, 42]	1941	F	44	Tumor and pain	3rd rib anterior	3 yr.	a. Resection of anterior 3rd rib b. Resection of recurrence with adjacent ribs and pleura	4 5	a. Recurrence at 2 yrs. b. Living and well 8 yrs. after 2nd operation	Chondrosarcoma
36. Ferrari and Lentino[13]	1942	M	--	Tumor	9th rib, right posterior axillary line	3 yr.	a. "Extirpation" b. Reoperated 1 yr. later c. "Extirpated" tumor with segment of 8th rib and pleura	1 1 5	a. Recurrence in few months b. Recurrence in a few months then slow growth for 16 yr. c. Living and well 19 mo.	Osteochondrosarcoma

37. Harper[22, 23]	1942	F	50	(Trauma) Pain	7th and 8th ribs, right, anterior	10 yr. (24 yr. after trauma)	a. Tumor "delivered;" portions of 7th & 8th ribs removed; diaphragm dissected off tumor b. More of chest wall removed. Implants on pericardial surface not removed	4 2	a. Recurrence at 8 yr. b. Living and "getting along very nicely" at 2 plus yr.	Chondrosarcoma
38. Sommer and Major[63]	1942	M	34	Tumor	8, 9, 10th ribs right, posterior	3 mo.	Exploration; not resectable	2	Dead 1 yr. Pulmonary and epidural metastases	Chondrosarcoma
39. Sommer and Major[63]	1942	F	24	Pain	6th rib, right axillary	5 mo.	6th and 7th ribs widely resected. Tumor removed from lung by sharp dissection	4	Dead; metastases 25 mo. postoperatively	Chondrosarcoma
40. Sommer and Major[63]	1942	M	38	Tumor	4th-10th ribs, posterior	8 yr.	a. Excised; no details b. Excision of axillary mass	1 2	a. Recurrence 3 mo. b. Dead; 2 mo.	Myxochondroma
41. Sommer and Major[63]	1942	M	49	Tumor	Gladiolus	5 mo.	Sternum resected; tumor freed from pleura and prepericardial fat by blunt dissection	4	Survived operation No follow-up*	Chondrosarcoma
42. Jaffe[33]	1943	M	--	Tumor	6th rib left, midaxillary line	2 yr.	a. Removed in several pieces b. Wider resection	2 4	a. Recurrence-4 mo. b. Local recurrence in few months	Chondrosarcoma
43. Pendergrass, Lafferty and Horn[50, 51]	1945	M	55	Tumor	7th rib, right midaxillary line	1 yr.	Resection	4	Living and well 7 yr.	Chondrosarcoma

TABLE I—CONT'D

AUTHOR	YEAR	SEX	AGE ONSET (YR.)	INITIAL SYMPTOMS	LOCATION	DURATION UNTIL FIRST TREATMENT	OPERATION	CATEGORY OF OPERATION	OUTCOME	AUTHOR'S DIAGNOSIS
44. Pendergrass, Lafferty and Horn[50, 51]	1945	F	39	None (X-ray discovery)	11th rib, left posterior (near angle)	1 yr.	Resection of tumor with 4 cm. segment of ribs	4	Living and well 6 yr.	Chondrosarcoma
45. Morton and Mider[45]	1947	F	27	Tumor	7, 8, 9th ribs	1 yr.	a. Removal of tumor with 4" piece of rib	4	a. Recurrence 15 mo.	Chondrosarcoma
							b. "Tumor removed"	1	b. Recurrence	
							c. Resection of ribs 7, 8, 9 and pleura	5	c. Recurrence 3 mo. Dead 6 yrs. after initial operation	
46. Watson and James[68]	1947	F	53	Tumor	9th and 10th ribs left anterior	2 yr.	Resection of portion of ribs 9-12 with portion of pleura, peritoneum and diaphragm	5	Died 18 days postoperatively. (Pulmonary embolism)	Chondrosarcoma, low grade
47. Dorner and Marcy[11]	1948	F	31	Tumor	11th rib, left posterior axillary line	20 yr.	a. "Mass peeled out readily;" pleura separated with some difficulty	3	a. Recurred at 9 yr.	Chondrosarcoma, low grade
							b. Recurrence in scar excised	3	b. —	
							c. Chondrosarcoma of mandible excised (probably new tumor)	-	c. Died postoperatively (12 yrs. after rib tumor first removed)	
48. Dorner and Marcy[11]	1948	M	53	Tumor	6th and 7th ribs lateral	1 yr.	a. Resection of tumor with 6th and 7th ribs	4	a. Recurrence 6 mo.	Chondrosarcoma
							b. Palliative partial removal	2	b. Dead 3 mo. (2 yr. 3 mo. after first operation)	

49. Dorner and Marcy[11]	1948	M	32	Pain and tumor	10th rib, left	1 yr.	Radical resection, including portion of diaphragm	5	Living and well 4 mo.	Osteochondromyxosarcoma
50. O'Neal and Ackerman	R. C. W. 28434	M	11	Tumor	Manubrium	12 yr.	a. Tumor removed with osteotome	3	a. Recurrence 1 yr.	Chondrosarcoma
							b. Removed piecemeal and chiselled off sternum	3	b. Recurrence 1 yr.	
							c. Recurrent nodule removed as outpatient	3	c. Recurrence 5 yr.	
							d. Tumor removed from sternum with osteotome "Daughter tumor pearls removed from adjacent muscle."	3	d. Recurrence	
							e. Entered with recurrence in manubrium and cartilaginous tumor of lumbar spine. Spine tumor partially removed	-	e. Dead 50 months of paraplegia (16 yr. 5 mo. after initial operation)	
51. O'Neal and Ackerman	C. D. M. 28578	M	48	Pain	Lower sternum	7 mo.	Lower sternum resected widely; obvious tumor left attached to pericardium	2	Dead 4 yr. 6 mo.	Chondrosarcoma
52. O'Neal and Ackerman	N. W. Mc. 42799	M	45	Pain	5th rib, right anterior	7 mo.	a. Portion of involved 5th rib removed along with some "cheesy" material behind	1	a. Recurrence 2 mo.	Chondrosarcoma
							b. Block resection of 4, 5, 6; overlying skin and pectoral muscle, underlying pleura	5	b. Postoperative empyema, recurrence in sinus tract 8 mo.	
							c. Partial removal of recurrent tumor masses	2	c. Dead 8 mo. (2 yr. 2 mo. after initial operation)	

TABLE I—CONT'D

AUTHOR	YEAR	SEX	AGE ONSET (YR.)	INITIAL SYMPTOMS	LOCATION	DURATION UNTIL FIRST TREATMENT	OPERATION	CATEGORY OF OPERATION	OUTCOME	AUTHOR'S DIAGNOSIS
53. O'Neal and Ackerman	A. M. 56540	F	68	Tumor	4th rib, right anterior	8 mo.	a. "Tumor removed" no details	1	a. Recurrence 1½ yr.	Chondrosarcoma
							b. Two-stage operation. Removal of tumor, adjacent ribs and adjacent pectoral muscles. Tumor dissected from pleura	4	b. Recurrence 1½ yr.	
							c. Tumor partially removed	2	c. Persisted	
							d. Large amounts of tumor "scooped out."	2	d. Dead—postoperative empyema. (4 yr. after initial operation)	
54. O'Neal and Ackerman	M. B. K. 56639	M	61	Tumor	3rd rib, left anterior	3 yr.	a. Removal of 3 inches of left 2, 3, and 4th ribs with a little pleura and lung tissues	5	a. Recurrence at 1 yr.	Chondrosarcoma
							b. Locally invasive to extent that removal impossible; greater portion of tumor removed.	2	b. Dead—7 mo. (2 yr. after initial operation)	
55. O'Neal and Ackerman	J. W. N. 92790	M	36	Pain	12th rib, left presenting retroperitoneally	3½ mo.	Removed with portions of 10, 11 and 12th ribs and portion of diaphragm. Tumor ruptured during removal and cyst contents extruded into wound	3	Recurrence at 6 mo. Died 11 mo.	Chondrosarcoma

56. O'Neal and Ackerman	C. P. 111305	F	25	Pain	7th rib, left posterior	6 mo.	Tumor partially removed	2	No follow-up	Chondrosarcoma
57. O'Neal and Ackerman	J. K. C. 55153	M	74	Tumor	9th rib, right anterior	--	Biopsy only	0	Died 22 mo. after onset	Chondrosarcoma
58. O'Neal and Ackerman	J. H. L. 141118	M	60	Tumor	10th rib, right anterior	6 mo.	Removal with half of involved 10th rib, overlying skin and muscle, underlying pleura, adjacent intercostal muscles and periosteum of adjacent ribs	5	Living and well 18 mo.	Chondrosarcoma
59. O'Neal and Ackerman	J. M. 180662	M	70	Pain	3rd rib, left posterior	3 mo.	Tumor partially removed. Not resectable because of involved subclavian vessels	2	Recent case	Chondrosarcoma
60. O'Neal and Ackerman	R. H.* G M & O 452	M	58	Tumor	9th rib, left anterior	6 yr.	Resected with adjacent 8th and 10th ribs, 3 inches of involved rib, pleura. Diaphragm incised in order to remove tumor.	5	Living 3 yr. 3 mo. Recurrence not evident	Chondrosarcoma

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*Reported by courtesy of Dr. James Mudd and Dr. John Saxton of Missouri Pacific Hospital, St. Louis, Mo.

TABLE I. SUMMARY OF CASES

Group II: So-Called Benign

AUTHOR	YEAR	SEX	AGE ONSET (YR.)	INITIAL SYMPTOMS	LOCATION	DURATION UNTIL FIRST TREATMENT	OPERATION	CATEGORY OF OPERATION	OUTCOME	AUTHOR'S DIAGNOSIS
61. Amburger[1]	1901	F	29	Tumor	3rd-6th ribs, anterior, right	1 yr.	Ribs 3, 4, 5 and 6 resected 1 cm. from tumor; portion attached to lung left behind; pleura and muscle removed	2	Dead—postoperative empyema 11 postoperative day	Enchondroma, with myxomatous parts
62. Gangolphe and Tixier[16]	1902	F	36	Tumor	Right sternoclavicular area (manubrium)	3 yr.	Resection of upper sternum, inner portions of both clavicles and first ribs. Pleura not removed, though attached	4	Survived operation. No follow-up	Enchondroma, perhaps becoming malignant
63. Richardson[56]	1913	M	26	Tumor	Manubrium	"Years" growth for 7 mo.	Manubrium resected, with portion of clavicles pectoral muscles, and 1st costal cartilages. Innominate vessels "eased" away from tumor	4	Survived operation. No follow-up	Enchondroma
64. Denk[10]	1930	F	28	Pain	Left posterior chest	2 yr.	Tumor resected with three ribs and parietal pleura and intercostal muscles	5	No follow-up	Chondroma
65. Morton[44]	1930	M	60	Pain	8th rib, right anterior	3 yr.	Resection of ribs 7 and 8 and attached cartilage. Separated from parietal pleura without damage to pleura	4	Living and well 1 yr. 2 mo.	Osteochondroma
66. Geschickter[17]	1931	M	24	Tumor	"Rib"	--	Excision	1	No follow-up	Chondroma

67. Geschickter[17]	1931	M	43	Tumor	Sternum	--	No operation	0	Living 11 yr. after onset with "signs of malignant change"	Chondroma
68. Follinsson and Blanchard[14]	1933	M	Birth	Tumor	6th rib, posterior right	3½ mo.	Removal with 6th and 7th ribs. Tumor removed piecemeal. Pleura not removed	3	No follow-up	Chondroma
69. Hedblom[28]	1933	F	60	Tumor	Lower sternum	--	a. 2-3 cm. tumor excised b. Recurrence removed c. Curette, sharp dissection, cautery	1 1 3	a. Recurrence 4 mo. b. Recurrence 3 mo. c. Recurrence 2 mo.	Chondroma
70. Hedblom[28]	1933	M	30	Tumor	2nd and 3rd ribs left anterior	8 mo.	Two-stage removal. Parietal pleura not removed	4	No follow-up	Osteochondroma
71. Potvin[55]	1933	M	26	Tumor	10th rib anterior	3 mo.	Removal with 10th rib	4	No follow-up	Chondroma
72. Sorrel and Oberthur[44]	1933	M	9	Tumor	2nd rib anterior, left	2 yr.	Partial removal because of adherence to great vessels and pericardium	2	Living and well 1 yr.	Chondroma "without actual evidence of malignancy"
73. Auchincloss[2]	1934	M	16	Tumor	8-9 ribs, right posterolateral	7 yr.	Two-stage removal of 7-10 ribs. Mass dissected off pleura and diaphragm	4	Dead—postoperatively	Osteochondroma. Identical bony nodule in lung thought not to be a metastasis.
74. Moura and Rodrigues[46]	1935	M	21	Tumor	1st rib posteriorly	4 mo.	Removal with first rib	4	Living and well 2 yr.	Osteochondroma
75. Sokolov[62]	1935	F	27	Tumor	8, 9, 10th ribs anteriorly	10 yr.	Removal, 8, 9, 10th ribs	4	No follow-up	Chondroma
76. Santy[60]	1937	M	34	Tumor	3rd and 4th ribs left, anteriorly	4 yr.	Removal with 3rd and 4th ribs en bloc, with intercostal muscles and parietal pleura	5	No follow-up	Chondroma

Table I—Cont'd

AUTHOR	YEAR	SEX	AGE ONSET (YR.)	INITIAL SYMPTOMS	LOCATION	DURATION UNTIL FIRST TREATMENT	OPERATION	CATEGORY OF OPERATION	OUTCOME	AUTHOR'S DIAGNOSIS
77. Golla[18]	1938	M	40	Tumor	3rd right, anterior	9 yr.	a. Removal with portion of 3rd and 4th ribs. Pleura not resected b. Resection of chest wall and pleura 3 yr. after recurrence	4 5	a. Recurrence 2 yr. b. Living and well 5 mo.	Chondroma
78. Harper[21, 23]	1939	F	16	Tumor	4th rib, left anterior	1 yr.	4th rib and portion of sternum removed; line of cleavage developed between tumor and pleura	4	Living and well 4 yr.	Chondroma
79. Head[25, 26]	1939	F	33	Tumor	Manubrium	2 yr.	Excision of manubrium	4	Living and well 11 yr.	Benign chondroma
80. Janes[34]	1939	M	30	None (X-ray discovery)	3rd rib, right anterior	0	1 inch of rib and 1 inch of costal cartilage removed away from tumor; underlying pleura removed	5	Living and well 1 yr.	Chondroma
81. Janes[34]	1939	M	38	Tumor	9th rib, left anterior	2 yr.	Removal with neighboring ribs, cartilage, soft tissue and pleura	5	Living and well 21 yr.	Benign osteochondroma
82. Bertola and Martinez[3]	1940	F	37	Tumor	4th rib, anteriorly	1 yr.	Operation	1	No follow-up	Osteochondroma
83. Janes[35]	1941	F	12	Tumor	Right anterior chest	3-4 yr.	a. "Lump removed" b. 20 yr. after onset tumor removed with adjacent sternum and chest wall	1 5	a. Recurred b. Well—time of follow-up not stated	Osteochondroma

84. Janes[35]	1941	M	51	Tumor	1st rib anteriorly	6 mo.	Removal of tumor and some sternum	4	Survived operation. No follow-up	Osteochondroma
85. Janes[35]	1941	F	64	Tumor	1st rib anteriorly, left	Tumor since childhood. Growth 3 yr.	Removal of tumor with adjacent rib and cartilage	4	No recurrence; time not stated	Benign chondroma
86. Janes[35]	1941	F	35	Tumor	Right anterior chest wall	No treatment	No operation	0	Died of infection in chest wall, tumor of 30 yr. duration	Chondroma (myxomatous degeneration)
87. Simpson[61]	1942	F	14	Horner's syndrome	1st rib, left posterior	20 yr.	Removal of 1st rib and portion of 2nd; pleura stripped away from tumor	4	Living and well 8 mo.	Osteochondroma
88. Sommer and Major[63]	1942	M	33	Pain	Gladiolus	10 mo.	a. Tumor curetted from sternum	3	a. Recurrence	Chondroma
							b. Tumor removed with gladiolus, portions of costal cartilages 3-7; dissected off pleura pericardium	4	b. Recurrence 8 mo.	
							c. Excision of nodule from sinus tract	3	c. Living and well 6 yrs. 10 mo. after last operation*	
89. Sommer and Major[63]	1942	M	47	Pain	7th rib, posteriorly left	3 mo.	6-9th ribs resected, tumor stripped from parietal pleura. Tumor invaded vertebral bodies and this left in	2	Living and well 5 yr.*	Chondroma
90. Sommer and Major[63]	1942	M	20	Tumor	Gladiolus and manubrium	6 mo.	Tumor removed. Bone of sternum removed to depth of 1 cm.	3	Living and well 4 yr. 2 mo.	Chondroma
91. Sommer and Major[63]	1942	F	54	Tumor	10th rib, left midaxillary	1 yr.	En masse removal, 9-11 ribs and pleura. Tumor dissected off peritoneum	4	Living and well 20 mo.	Osteochondroma

TABLE I—CONT'D

AUTHOR	YEAR	SEX	AGE ONSET (YR.)	INITIAL SYMPTOMS	LOCATION	DURATION UNTIL FIRST TREATMENT	OPERATION	CATEGORY OF OPERATION	OUTCOME	AUTHOR'S DIAGNOSIS
92. Santy, Dargent and Marion[60]	1946	M	50	Tumor	Lower sternum (Xiphoid)	12 yr.	Resection of costal cartilages 4-5 cm. away from tumor. Amputation of sternum. Pleura and pericardium not removed	4	Living and well 1½ mo.	Chondroma
93. Morton and Mider[45]	1947	F	45	Tumor	3rd and 4th ribs, right, anterior	5 yr.	Removal of tumor and portions of 3rd and 4th ribs	4	No recurrence locally; at age 57 patient developed symptoms of tumor of right ilium, which proved to be chondrosarcoma. Died at 64. Recurrence in ilium and pulmonary metastases	Benign chondroma
94. Watson and James[68]	1947	M	18	None (discovered on physical exam.)	4th rib, left, anteriorly	2 mo.	a. Resection of 4th rib b. Removed with portions of ribs 3 and 4; pleura resected (7 mo. after first operation)	4 5	a. Recurrence 4 mo. in rib stump b. No follow-up	Chondroma
95. Dorner and Marcy[11]	1948	F	18	Tumor	2nd rib, left anterior	15 yr.	Excision of tumor, adjacent cartilage and rib, pleura	5	Living and well 15 mo.	Osteochondroma
96. Poppe and Berg[54]	1948	M	55	Tumor	6th rib, right anterior	10 yr.	Removal of segments of ribs 5-7 and pleura underlying	5	No follow-up	Osteochondroma

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*After preparation of this paper was completed, Dr. Cameron Haight of Ann Arbor, Michigan, informed us that Sommer and Major's cases No. 2, 3, and 9 (Cases 88, 89, and 41 respectively in Table I) were living and apparently well at 18, 15, and 10 years following operation.

Sex: Males predominated in both groups. In the chondrosarcoma cases, there were thirty-nine males and twenty females. The sex of one patient was not stated. Of the chondroma and osteochondroma cases there were twenty-one males and fifteen females (65.0 per cent males in Group I and 58.3 per cent males in Group II).

Symptoms: In forty (66.7 per cent) of the patients in Group I, the first symptom was the presence of a tumor mass. In sixteen (26.7 per cent) the first symptom was pain, usually of three types: (1) local pain—dull and aching—at the site where tumor eventually appeared, (2) pleuritic pain which occurred *chiefly in cases where the bulk of the growth was intrathoracic,* and (3) neuritic pain referred along the distribution of an intercostal nerve. This neuritic pain caused confusion in one of our cases (J.W.N.) in which pain from a tumor of the twelfth rib was referred to the groin and was thought at first to be renal in origin. Some of the reported cases had pain in the right upper quadrant of the abdomen and were originally believed to have gall bladder disease. Pain was more frequent as an initial symptom in the malignant group where it occurred in 26.7 per cent than in Group II where it was present in 11.1 per cent. Most *of the cases which had pain as the initial symptom developed a palpable tumor* later, and many of those in which the initial indication of disease was the appearance of a mass developed pain as a later symptom. Three cases presented no symptoms, but tumor was discovered on routine physical examination or chest roentgenograms. Simpson[61] and Denk[10] have reported cases in whom Horner's syndrome was present. Many cases, of course, developed dyspnea, ulceration, infection in the chest wall, and even empyema, but these are late phenomena and need not be considered in the early case.

There was no essential difference between Group I and Group II in the duration of symptoms before the first operation (Table II). While about half of the patients presented themselves for treatment in the first two years, more than 10 per cent in both groups had symptoms of ten or more years' duration (Table II).

Physical Examination.—On examination, the patients generally presented a hard, sometimes nodular tumor that was fixed to the bony chest wall (Fig. 3). Occasionally soft fluctuant areas could be felt, particularly in the larger tumors.

Location.—Hedblom[27, 28] and Heuer[29, 30] found that about 80 per cent of all the tumors of the bony thorax arose in the ribs, and 20 per cent in the sternum. In the present series of cartilaginous tumors, eighty (83.3 per cent) arose in rib and sixteen (16.7 per cent) arose in the sternum. There are no significant differences between Group I and Group II regarding site of origin. *Any rib may be involved* without preference for either the upper or the lower ribs. There is a predisposition for these tumors to originate in the rib near the costochondral junction. Of the eighty rib tumors, eleven were not specifically localized. Forty-seven (68.1 per cent) of the remaining sixty-nine tumors were located near the costochondral junction, eighteen (26.1 per cent) were located posteriorly, and four (5.8 per cent) at other points in the shaft of the rib. Of the tumors arising posteriorly, most seemed to originate at the angle, and some in the head of the rib.

TABLE II. SUMMARY OF CLINICAL FEATURES

CLINICAL FEATURES	GROUP I 60 CASES	GROUP II 36 CASES
Age at onset of symptoms		
Mean	41.4 years	32.6 years
Limits	11-74 years	Birth-60 years
Sex		
Male	39 (65.0%)	21 (58.3%)
Female	20 (33.3%)	15 (41.7%)
Not stated	1	--
Initial symptoms		
Tumor	40 (66.6%)	29 (80.6%)
Pain	16 (26.7%)	4 (11.1%)
Other	4 (6.7%)	3 (8.3%)
Location		28 (77.8%)
Rib	52 (86.7%)	8 (22.2%)
Sternum	8 (13.3%)	
Duration of symptoms until first operation		
2 years or less	34 (56.7%)	18 (50.0%)
10 years or more	7 (11.7%)	5 (13.9%)

Fig. 3.—Patient with a chondrosarcoma of the manubrium.

Most of the sternal tumors were not further localized, but location in the manubrium was slightly more frequent than in the gladiolus. Origin in the xiphoid seems to be rare.

X-ray Examination.—Pendergrass, Lafferty, and Horn[31] have pointed out that the pathologic type of malignant bone tumor cannot be diagnosed with

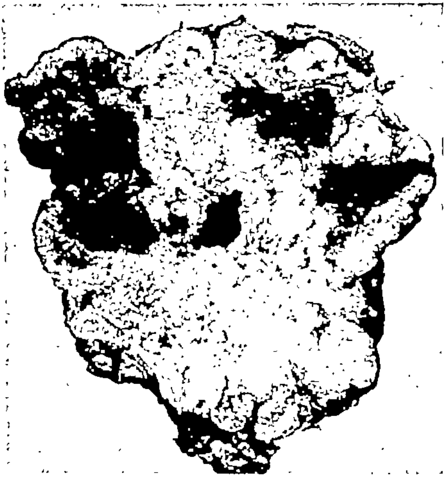

FIG. 4A.—Surgical specimen (cut surface) of a 15 cm. chondrosarcoma of the 12th rib, showing calcification and cyst formation.

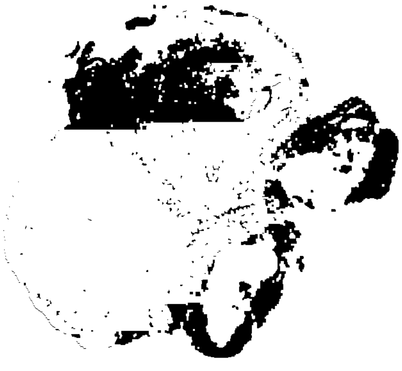

Fig. 4B.—X-ray of specimen shown in Figure 4A.

certainty from roentgen examination alone. Although they found a characteristic roentgen pattern for sclerosing and osteolytic osteogenic sarcoma and for central and peripheral chondrosarcoma, there were cases that were not typical roentgenographically. The distinguishing feature of peripheral chondrosarcomas is the presence of large masses arising from bone with irregular spotty flecks of calcification (Fig. 4). The central chondrosarcomas are demonstrated as thin-walled cavities near the ends of long bones. Irregular scattered foci of calcification may be seen in the radiolucent cavity. When the thinned-out cortex is perforated, there may be "sun-ray" spicules of bone arising from the surface of the bone.

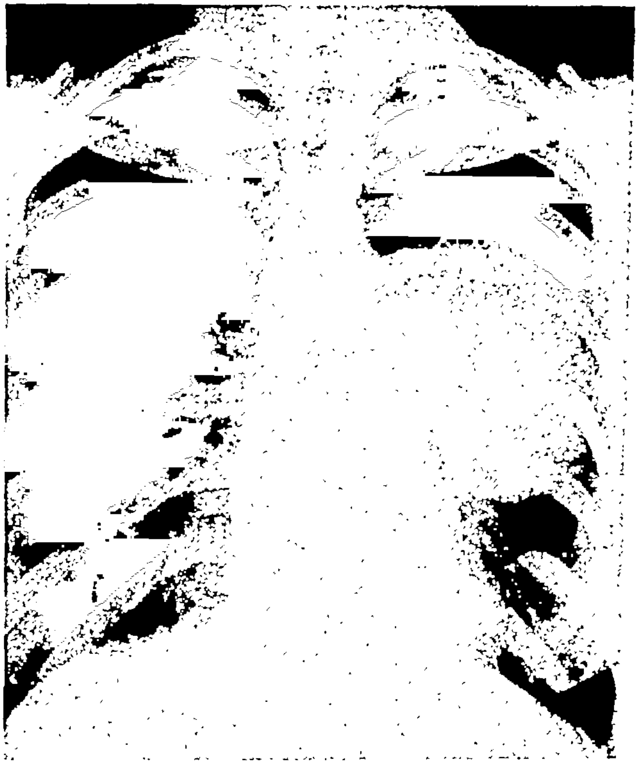

Fig. 5.—Chondrosarcoma of rib presenting in the posterior mediastinum. Note destruction of the posterior portion of the 7th rib.

The majority of our cases were recognized on x-ray examination as being primary tumors of rib or sternum, and the majority were termed of cartilaginous origin, although one was called a periosteal fibrosarcoma and two were called osteogenic sarcoma. The chief error in interpretation was in calling malignant cartilaginous tumors benign. This error is probably unavoidable in many cases. The two tumors in our series arising from the posterior portion of a rib developed in the posterior mediastinum and were considered malignant neurofibromas invading rib (Fig. 5). Although the usual metastatic carcinoma to bone is osteolytic, confusion may be expected occasionally from osteosclerotic secondary neoplasms. Osteolytic metastases may sometimes simulate enchondromas.

Multiple Tumors.—Six instances (Cases 14, 25, 43, 47, 50, 93) of multiple cartilaginous tumor appear in this series. The incidence of tumors of the ribs and sternum in patients with multiple hereditary osteochondroma is high, and these six cases do not reflect the true incidence, but only those in whom the tumor was predominant clinically. Five of the six had malignant chest wall tumors, and two (Cases 47 and 93), and possibly one other (Case 50), developed a new primary chondrosarcoma at another site. Obviously, all of these tumors cannot be removed, but close observation is certainly indicated.

Differential Diagnosis.—Ordinarily the diagnosis of a cartilaginous tumor of rib or sternum should not be difficult to make on the basis of history, physical examination, and roentgen examination; but roentgen examination may not be absolutely diagnostic, and there is often great difficulty in obtaining adequate roentgen views of these tumors, particularly small tumors of the sternum and the extreme anterior ends of ribs. Fine flecks of calcium, important in differential diagnosis, may not be visualized. Other primary bone tumors as well as metastatic tumors must also be considered in the differential diagnosis. Cold abscesses and aneurysms of the great vessels eroding the chest wall may appear as firm tumors and cause confusion temporarily. At Barnes Hospital the most common non-neoplastic condition misdiagnosed as chondroma or osteochondroma was the effect of trauma. In two instances, malunited costochondral separations were resected, and in another case, a malunited fracture of a costal cartilage was resected. In yet another case, a large anteflexed but otherwise normal xiphoid was operated on with the preoperative diagnosis of osteochondroma. Each of these patients presented himself, three of them years after local trauma, because of a "tumor" which was believed to be slowly enlarging. Roentgenograms were obtained in each of these four instances, but the areas in question were not sufficiently well visualized to allow the diagnosis of primary bone tumor to be excluded. Neurilemmomas and neurofibromas of intercostal nerves, especially those growing anteriorly, may simulate primary rib tumor. We have seen such a case recently, and Freedlander[15] has called attention to the possibility of misdiagnosis in this circumstance. Low grade osteomyelitis has also been mistaken clinically for rib tumor.[31] Bronchiogenic carcinoma invading the chest wall should be considered in the differential diagnosis. Posterior mediastinal tumors of all varieties may cause diagnostic uncertainty, as well as teratomas, thymomas, and substernal thyroid lesions anteriorly, particularly if they exhibit spotty calcification, as they occasionally do.

If there is doubt as to the diagnosis, it may be necessary to resort to biopsy. Either aspiration or incisional biopsy should confirm the cartilaginous nature of the tumor, but will not necessarily determine whether the tumor is benign or malignant. Thoracoscopy[39] and artificial pneumothorax have been used as diagnostic measures but are probably unnecessary in most cases.

Gross Pathology.—It is possible to determine the cartilaginous origin in most cases on gross examination but, as we have mentioned above, whether or not the tumor is benign or malignant cannot be assessed with certainty. The osteochondromas, so-called, are exophytic lesions in which the bone component is not distributed in a disorganized fashion but arranged in lines roughly perpendicular to the portion of bone from which it arises. With marked enlargement of the tumor this pattern becomes greatly distorted. The neoplastic portion of this tumor is the cartilaginous cap. Enchondromas occur in ribs and sternum and present there the same picture as elsewhere. The cortex is thinned and expanded at the site of the neoplasm, and the medullary cavity is occupied by the bluish or pearl gray nodular tumor.

It is not possible in many cases to determine whether the tumor began as an enchondromatous or ecchondromatous lesion, because the portion of the rib of origin is often completely destroyed. Examples of both types may be seen in ribs and sternum. The tumors may reach tremendous size: the largest in our series was 15 cm. in greatest diameter, and tumors 30 cm. in greatest dimension[22] and the "size of a child's head"[10] have been reported. The grapefruit, the orange, and the lemon have been common standards of measurement. The larger tumors may contain cystic areas of degeneration with the cysts filled with viscid mucoid material which may be clear, turbid, or stained with altered blood. Often, too, the cysts contain material which has the appearance and the consistency of cooked rice.

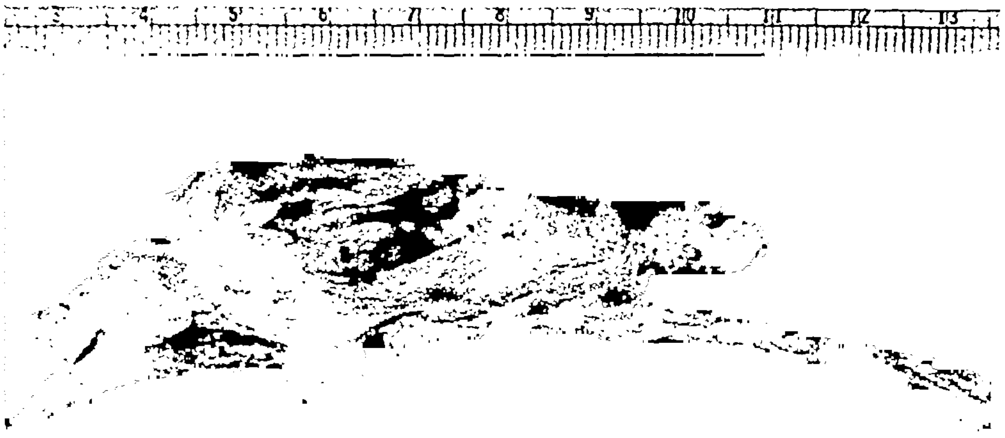

Fig. 6.—An ecchondromatous chondrosarcoma of the 10th rib near the costochondral junction. The "satellite" nodules are pedunculated extensions of the main mass.

Most of the tumors, even the malignant ones, are encapsulated to some degree. The portion of the tumor growing in soft tissue can frequently be "shelled out" of its capsule or pseudocapsule. This encapsulation is a treacherous characteristic, since in enucleating the tumor, satellite nodules or portions of the malignant perichondrium may be left behind. Satellite nodules are more apparent than real inasmuch as if the tumor has not been previously treated, a connection between nodule and main tumor may be demonstrated. The satellite nodule represents a local growth acceleration of a nodule at the periphery of the main tumor (Fig. 6).

The nodularity of these tumors, and the mosaic pattern seen on cut surface occasioned by the nodularity, is often striking (Fig. 7). The nodules themselves are cartilaginous and are separated by thin fibrous tissue septa. The beginnings of cystic degeneration may be seen in the center of some of the nodules. In the less malignant tumors, the center of the nodules may contain calcific material or even bone.

Adjacent structures may be invaded by tumor and this fact not recognized if the invasion is present to only a slight or moderate degree. The advancing margin of the tumor, even of the most malignant, is discrete, and the adjacent soft tissue (pleura, pericardium, diaphragm, or skeletal muscle) is first displaced, distorted, and then invaded. It is often impossible to make the differentiation between marked distortion and slight invasion. Recurrent tumors exhibit the same gross characteristics as the primary tumors but may not be attached to bone and usually contain less new bone and calcific material.

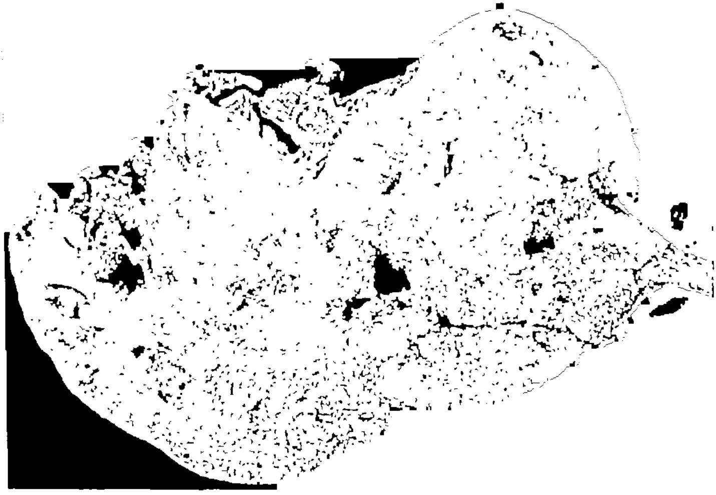

Fig. 7.—Cut surface of surgical specimen (chondrosarcoma of 9th rib) showing the characteristic glistening pearly appearance and the gross nodularity.

Microscopic Pathology.—Microscopically, the osteochondroma appears to develop by enchondral bone formation which does not differ a great deal from the normal growth of a long bone except that it is less orderly. The differentiation between an osteochondroma and a chondroma is largely a gross differentiation, for bone formation simulating enchondral osteogenesis is seen, but to a lesser degree, microscopically in most chondromas. The slightly or moderately malignant chondrosarcomas also sometimes exhibit this phenomenon, but in a lessened degree and a more haphazard manner (Fig. 8). In general, the more malignant the tumor, by other criteria, the less the bone formation. The stroma may be quite varied in individual tumors and even in different areas of the same tumor. A homogeneous, hyaline, basophilic stroma is most characteristic, but in softened degenerated areas the stroma may be loose, finely granular, and chromophobic. Areas of acidophilic fibrous stroma are uncommon, but when found simulate a fibrosarcoma. Differentiation is possible with multiple sections, other areas demonstrating chondrosarcoma.

The appearance of the cells themselves, rather than the pattern of the tumor or the character of the stroma, is the most important factor in assessing the degree of malignancy. We accept the criteria of Lichtenstein and Jaffe[37] as stated previously; the benign tumors having regular cells with regular nuclei in the active portions, and the malignant tumors having varying degrees of nuclear atypism from occasional plump nuclei and double nuclei to large numbers of such nuclei and malignant giant cartilage cells (Fig. 9). Mitotic figures are not common in cartilaginous tumors, but were seen in two (20 per cent) of our cases (Fig. 10). Division by amitosis is the rule. Nuclear atypism is a better criterion of malignancy than the cellularity of the tumor. For instance, a tumor with the cells widely separate in a hyaline stroma but with a high incidence of atypical changes may be more malignant than one in which there is little stroma between the cells and a lower proportion of abnormal nuclei. The individual cells may be lacunated in hyaline stroma, stellate in mucinous stroma, or spindle shaped.

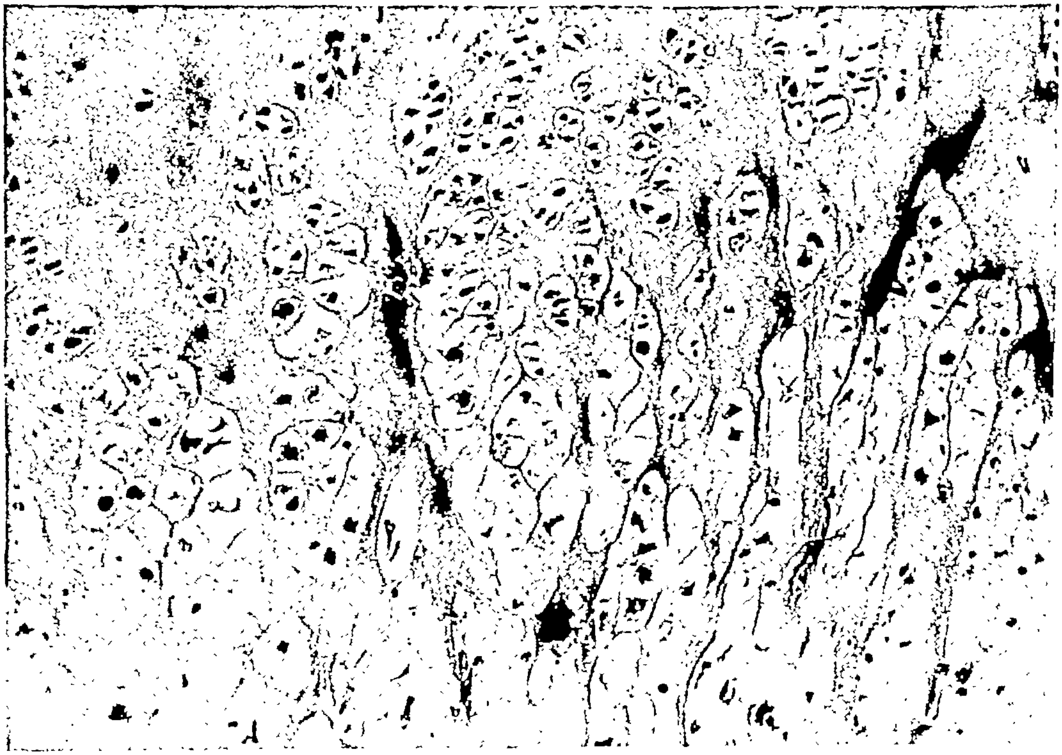

Fig. 8.—A pattern simulating enchondral osteogenesis in a sternal chondrosarcoma of low grade malignancy.

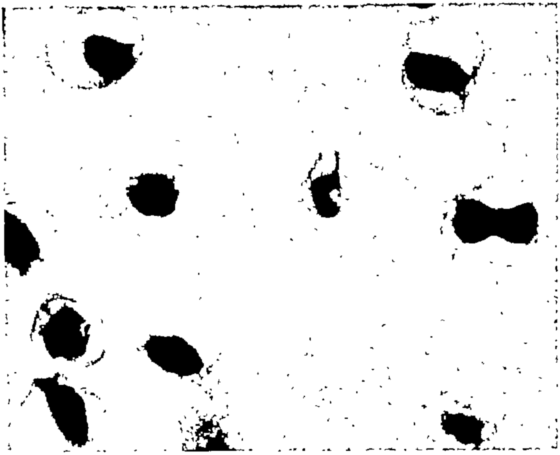

Fig. 9.—Photomicrograph showing a cell with two nuclei. When double nuclei are present in more than occasional numbers, a diagnosis of chondrosarcoma can be made.

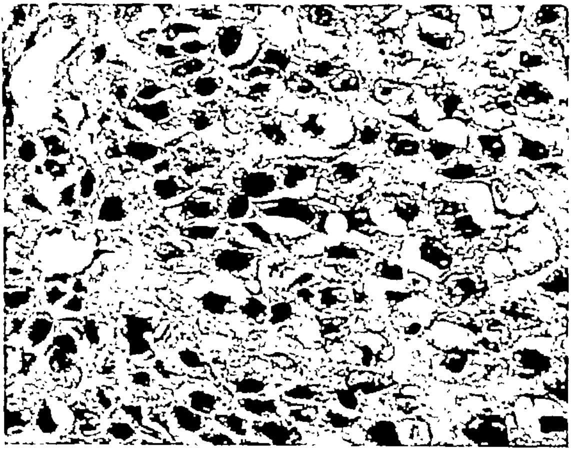

Fig. 10.—Mitotic figures in a chondrosarcoma. Usually this finding indicates a highly malignant tumor.

In a number of tumors in our series, the incidence of atypical changes varied from one field to another. In one tumor (J.H.I.) malignant change could not be identified in the bulk of the tumor, but sections from the edge showed definite chondrosarcoma. Lichtenstein and Jaffe[37] also noted this phenomenon of varying malignancy in the same tumor and emphasized the hazards in pathologic interpretation, particularly of small biopsy specimens.

The terminology applied to these tumors, chiefly on the basis of microscopic appearance, has been unnecessarily complex. In the tumors designated as myxochondroma and chondromyxosarcoma the addition of the qualifying *myxo-* adds little to the diagnosis, since the mucoid substance is not neoplastic but the product of degeneration. The nuclei in mucoid areas tend to be pyknotic and fragmented. Similarly, the designation osteochondrosarcoma is unwarranted since the bone in these tumors is not neoplastic but the stable product of a process resembling enchondral bone formation. All cartilaginous tumors should and can be designated as either chondroma or chondrosarcoma for purposes of simplification. The term osteochondroma is, however, well established by long usage, but it should be kept in mind that the neoplastic portion is the advancing cartilaginous cap and not the underlying bone, though the bony portion may comprise the bulk of the tumor. The osteoma may be an osteochondroma in which the cartilaginous cap has atrophied and disappeared.

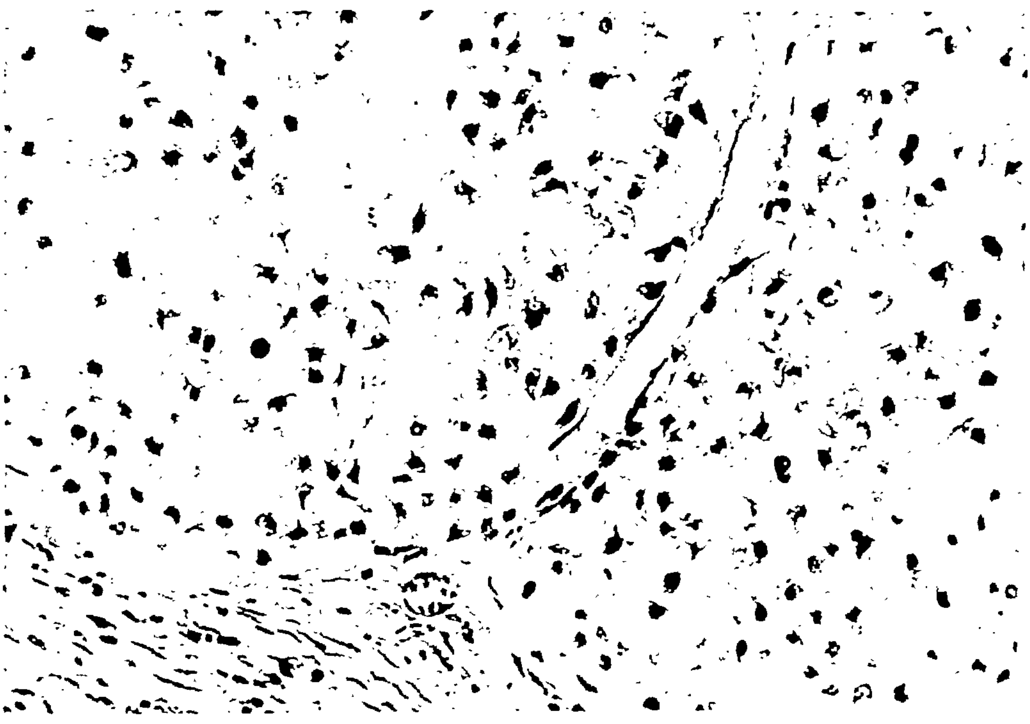

Fig. 11.—Photomicrograph of a moderately malignant chondrosarcoma. Note the variation in nuclear size.

Treatment.—Of the ninety-six cases reported and reviewed here, four were not treated because of advanced disease or refusal by the patient to be operated upon. An attempt has been made to classify the treatment of the remaining ninety-two cases into five categories, according to the "radicalness" of the operation. In a few cases, insufficient information was given to allow classification. The categories are:

(1) Insufficient information for classification
(2) Obviously inadequate removal—tumor partially removed
(3) Removal—Limited operations such as curretting, "shelling out," chiselling tumor off bone, tumor removed piecemeal, tumor ruptured with spilling of contents into wound.
(4) Resection—Excision of tumor and segment of bone of origin, with varying, but limited, amounts of adjacent soft tissue.
(5) Radical resection—Excision of tumor with segment of rib of origin, adjacent ribs, underlying pleura, and overlying skeletal muscle en bloc. This is a minimum and further structures may be included in the excision as indicated by the extent of the tumor.

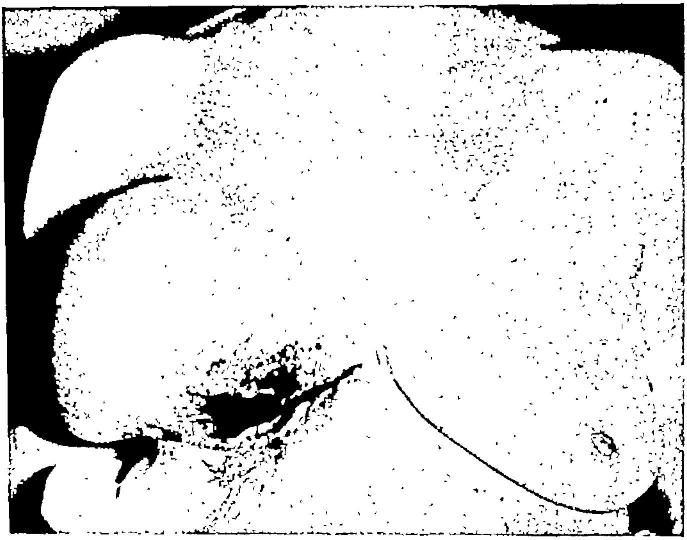

Fig. 12.—Recurrent chondrosarcoma of the 5th rib invading pectoral muscle, breast, and skin following two inadequate operations.

From the reports in the literature, the category of operation has been assigned as nearly as possible (Table I). We freely admit the possibility of occasional error. Categories 1 and 2 are self-explanatory. Concerning category 3, violations of the capsule or pseudocapsule which took place in "shelling out," removing piecemeal, or in accidentally rupturing the tumor, are included because of the almost uniformly poor results following these procedures in spite of wide resection elsewhere (Fig. 12). Seeding of the wound with tumor cells is suggested. Cases in category 4 often had wide resection of the chest wall with limited depth to the excision. Because the pleura is in such intimate proximity to the involved rib, it is often invaded early and must be removed with the block dissection. As early as 1897, Paget[48] pointed out the desirability of being prepared to enter the pleural cavity and of removing pleura, portions of adherent lung, pericardium, and diaphragm when dealing with cartilaginous tumors.

We feel it is important to remove rather long segments of rib. Although Upshaw and his associates[66] found that chondrosarcomas do not usually exhibit intramedullary extension for more than three inches in the involved bone, recurrences in the rib stump seem to be fairly common. That marrow extension is not the only reason for removing long portions of rib was shown by the case of Halpert and Davis[20] in which microscopic nodules of tumor were found in the periosteum distant from the tumor. The intercostal muscles above and below the tumor should also be removed, a procedure executed with least danger of transecting tumor nodules by removing segments of adjacent ribs.

Others[34, 68] have stressed that radical resection for malignant tumors of rib should include the structures outlined in category 5. Of course, the diagnosis of malignancy should be assured before undertaking such a mutilating operation, but in view of the fact that tumors of cartilaginous origin are either actually or potentially malignant, and that the malignant and benign cannot be distinguished with certainty on clinical grounds or on the basis of a single biopsy, we advocate radical resection for all monostotic cartilaginous tumors arising in ribs and sternum. Radical resection of the chest wall can be done with little deformity or loss of function compared to radical resection of a lesion of the humerus or femur which means the loss of an extremity. Rib is expendable. Jaffe and Lichtenstein[33] reported uniformly good results with curettement of solitary benign enchondromas of bone, but their cases were largely lesions of the bones of the hands and feet where the propensity to malignancy appears to be less than in the chest wall. It is not recommended that curettement be done for apparently benign enchondromas of rib or sternum. Wide resection of the chest wall should add but little to operative mortality and morbidity, but a great deal to insurance against recurrence.

Adequate radical resection cannot usually be carried out for those tumors invading deep into the mediastinum or into vertebrae, but the caprice of individual tumors may allow an occasional cure even under these circumstances if resection is as radical as possible. Invasion of lung does not contraindicate resection; portions of involved lung have been[30, 47, 48] and should be included in the block resection since wide local invasion may occur in the absence of distant metastases. Indeed, even before 1894[48] portions of involved lung were removed along with the chest wall at least six times.

The knowledge of repairing large defects in the chest wall will help insure more liberal radical resection. Gangolphe and Tixier[16] in 1909 first suggested the use of a metallic prosthesis to replace the resected sternum, but this and similar devices have not been widely used. To repair a very wide resection of the sternum, Kinsella, White, and Koucky[36] fixed long tibial grafts in the opera-

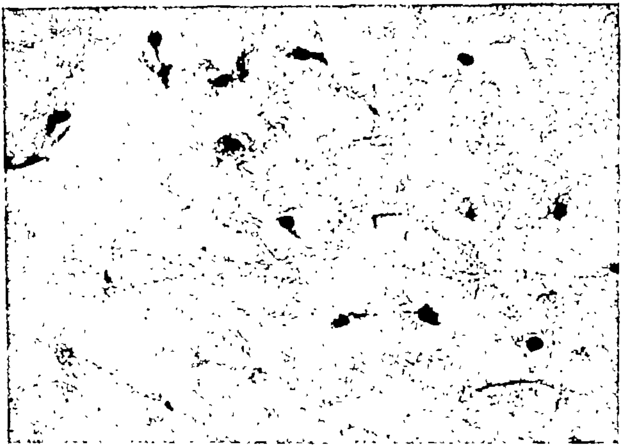

Fig. 13A.—A primary cartilaginous tumor of femur. The entire tumor was relatively acellular. A diagnosis of chondrosarcoma was made only by demonstrating a moderate number of atypical nuclear changes.

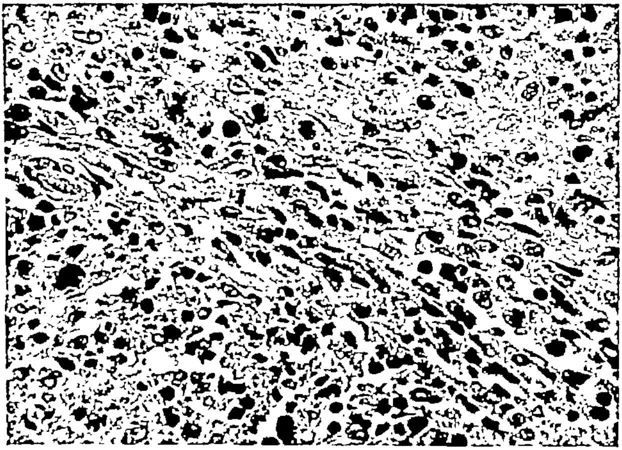

Fig. 13B.—A pleural metastasis from the tumor shown in 13A. Note the increased cellularity and the pattern suggesting a fibrosarcoma.

tive defect with resulting good stability. Bisgard and Swenson[4] suggest mortised rib grafts for the same purpose, but usually such types of repair are not needed. Head[25] resected the manubrium without replacement with no resulting deformity. Richardson[56] and Bradshaw and Chodoff[5] successfully resected the manubrium, portions of both clavicles, and both first costal cartilages without considerable deformity. Graham[19] removed all but the upper half of the manubrium, dividing all costal cartilages bilaterally except the first (Case C.D.M.) without resultant disability. Heuer[29] found the removal of the lower two-thirds of the sternum to be functionally "a minor matter."

Following chest wall resections for tumors of rib, Watson and James[68] used fascia lata to assist in the repair of the defect in four cases, with resulting good stability and lack of herniation of the lung in all. After a large resection of the chest wall, Pickrell and co-workers[53] successfully placed a split thickness skin graft on the pericardium with no incapacitation to the patient. Janes[34] divided the ribs adjacent to the defect and used these as a pedicled bone graft to partially bridge the gap. Maurer and Blades[40] concluded from their experience with defects of the chest wall occasioned by war trauma that "any defect of the chest wall, regardless of size, can usually be repaired by plastic procedures involving only structures which are part of the chest wall, namely rib, periosteum, and muscle." They considered the most important feature of the large repair to be the covering of the defect with bone or with periosteum which would form bone.

The only circumstance in which immediate operation is not recommended for chest wall tumors of cartilaginous origin is for the case of multiple cartilaginous tumors in which one or more of the tumors are located in rib or sternum. These tumors are usually first evident in childhood; after adolescence the rate of growth of the benign exostoses slows down, and during adult life no new ones form.[32] It would seem rational to observe these patients closely and to operate on only those tumors that continue to grow, grow at an increased rate, or appear after adolescence: that is, on the tumors most apt to be malignant.

Roentgentherapy has no place in the primary treatment of chest wall chondrosarcomas, and generally achieves little or nothing when administered for palliation.

Results of Operation.—Tables III and IV detail the results of operation in all cases. Four patients in both groups were not operated. There were 143

TABLE III. OUTCOME OF 102 OPERATIONS ON 58 PATIENTS*
(GROUP I CASES)

	OPERATIONS	DIED OF DISEASE DIED OF OPERATION (5 CASES) LIVING WITH RECURRENCE	LIVING AND WELL MORE THAN 2 YR.	NO FOLLOW-UP OR FOLLOW-UP LESS THAN 2 YR.
Category 1	24	22	1	1
Category 2	20	16	1	3
Category 3	18	16	0	2
Category 4	24	16	2	6
Category 5	16	8	3	5
Totals	102	78	7	17

*Two cases not operated.

TABLE IV. OUTCOME OF 41 OPERATIONS ON 34 PATIENTS*
(GROUP II CASES)

	OPERATION	DIED OF DISEASE DIED OF OPERATION (2 CASES) LIVING WITH RECURRENCE	LIVING AND WELL MORE THAN 2 YR.	NO FOLLOW-UP OR FOLLOW-UP LESS THAN 2 YR.
Category 1	5	3	0	2
Category 2	3	1	1	1
Category 3	5	2	2	1
Category 4	20	4	4	12
Category 5	8	0	1	7
Totals	41	10	8	23

*Two cases not operated.

operations in 92 patients exclusive of biopsies. It is impossible to determine "cure" rates because of the lack of adequate follow-up in many cases.

In operative categories 3, 4, and 5 the duration of time until recurrence was stated in thirty-two cases: in twenty-six (81.2 per cent), recurrences were noted in the first two years, and of the remaining six, recurrences appeared between two and five years in three, and three patients (9.4 per cent) were apparently well and without evidence of recurrence until eight, nine, and nine years following operation. Therefore, a *minimum* follow-up period of two years is necessary to determine outcome in most cases, while follow-up for life is probably necessary to determine cure rates for the entire group. Nineteen of *sixty cases (31.7 per cent) in Group I, and twenty-three of thirty-four cases* (67.7 per cent) in Group II were followed less than two years. It is very probable many of the Group II lesions have since recurred and displayed themselves as overt chondrosarcoma. Greater restraint in reporting inadequately followed cases is recommended.

Of the cases reviewed and reported, thirteen patients were living without evidence of recurrence for more than two years following attempted curative (Category 3, 4, and 5) operation. Six of these thirteen tumors (Cases 11, 16, 35, 43, 44, and 60) were chondrosarcomas (10 per cent of the 60 chondrosarcoma cases); and seven (Cases 74, 78, 79, 81, 88, 90, and 93) were classed as benign (19.4 per cent of the 36 Group II cases). As shown in Tables III and IV, most of these survivors had the benefit of Category 4 and 5 operations. It is significant that three of the apparently cured patients (11, 35, and 88) were operated for recurrence and were alive and well nine years, eight years and six years and ten months, respectively, following the second operation.

Two additional patients (Cases 13 and 89) were reported living and well for nine years and five years following inadequate (Category 2) operation. This is inexplicable except by assuming that judgment about the operation was incorrect or that quiescent or slowly growing tumor still lurks undetected by clinical examination.

There were no outstanding clinical differences regarding sex, duration of *symptoms, or location between the thirteen survivors and the group as a whole* (10 rib, 3 sternum). The age at onset of symptoms averaged about four years younger than that of the entire group.

EVOLUTION

Natural History.—It seems certain that chondrosarcomas may arise from pre-existing chondromas and osteochondromas, although the majority are clinically malignant from the start. The development of the tumor from benign to malignant has been traced pathologically.[33] The tendency to underdiagnose these tumors has made the evaluation of this point very difficult. Furthermore, although Harper[21] has emphasized the impossibility of determining clinically when a benign tumor changes into a malignant tumor, in most of the cases in which chondrosarcoma was thought to arise in a pre-existing benign tumor, the basis of the assumption was on clinical grounds. That is, a tumor present for many years with a steady slow rate of growth began to enlarge more rapidly, and malignant change was thought to have taken place. Many cartilaginous tumors develop in this manner, and perhaps the increase in growth rate is a logarithmic expression of the growth pattern of a tumor actually malignant from the start. Even those cases of chondrosarcoma in which an earlier microscopic diagnosis of benign tumor was made cannot always be used to prove malignant change, because the all important nuclear atypism may be overlooked or improperly evaluated. Some tumors demonstrate varying degrees of differentiation, and sections may not include the more malignant part. Three of our tumors were originally called chondroma, but on review of the original slides it was evident that chondrosarcoma was present at that time. Recurrences often appear histologically more malignant than the initial tumor,[33, 37, 45] but this phenomenon is not a consistent finding. We examined tissue from four patients on two or more occasions over periods from two to nine years. In one instance the histologic degree of malignancy increased, and in the remaining three the recurrences were histologically identical to at least parts of the original tumor.

Metastases are uncommon and occur as a late phenomenon and then often after several local recurrences. The metastases may appear more malignant than the primary tumor and even resemble a fibrosarcoma (Fig. 13A and B). In only one of our eleven cases was a metastasis to the lung evident on initial examination. Metastases are usually hematogenous, but may be due to seeding of the pleura, as Churchill[9] suggested regarding Case 28 in which nodules in the pleura appeared following a lobectomy for metastatic chondrosarcoma. The lung is the commonest site of hematogenous metastases; metastases to the epidural space ranks second in the reported cases, suggesting extension via the intercostal veins to the vertebral veins.

Roberg[57] concluded that chondrosarcomas of the distal portions of the extremities were inherently more favorable lesions than those more proximally located, and that as the site of the tumor approached the axis the malignancy was greater. We do not necessarily agree that this is a biologic phenomenon of chondrosarcomas. The more unfavorable prognosis of a chondrosarcoma as it approaches the axis can be partially explained by the increasing technical difficulties of its surgical removal and the greater reluctance on the part of the surgeon to perform a radical procedure. Further indication that Roberg's thesis may be correct, however, is found in Jaffe's[32] report of patients with multiple hereditary exostoses. In his three cases (out of 28 patients who had multiple tumors) that developed chondrosarcoma, the malignant tumors were located in a relatively central position: ilium, rib and sacrum. Confirmation of this point awaits a study of long term follow-up in many patients with cartilaginous tumors of all sites.

It has often been said[21, 57] that an incomplete operation greatly accelerates the rate of growth of a chondrosarcoma, but we found that many in this series had exhibited an increasing rate of growth before operation. Thirty patients who survived operation but were not cured of disease were followed until death or for long periods while living with recurrence. Eighteen of these (60 per cent) were *dead within the fifth year after the onset of symptoms and seven* (23.3 per cent) lived for ten years or longer, two of whom lived *with persistent chondrosarcoma* for twenty-eight years.

Prognosis.—The evaluation of prognosis entails the consideration of many factors. Chief among these are the type of operation and the pathologic nature of the tumor. In certain of our cases dying of disease, it is possible in retrospect to assess certain pathologic features which are of prognostic significance. We realize the hazards of such conclusions in a small series and appreciate that such prognostic assessment is only approximate. Table V illustrates this.

The presence of mitoses is the gravest sign and is associated with rapid growth and extension; in our small series the two cases presenting mitotic figures were *the only two in which evidence of distant metastases was found either* initially or later. The life span of the patient is also directly proportionate, in a fair number of cases, to the incidence of plump nuclei and double nuclei. The presence of bone, especially demonstration of a simulation of enchondral bone formation, implies some degree of differentiation. Actually the patients who demonstrated this lived longer even though uncured. In this respect it should be remembered that the tumor must be judged by its most malignant part, and the finding of an area of tumor cartilage forming bone by enchondral bone formation is not compatible with a good prognosis if another area is highly malignant in its microscopic appearance.

TABLE V. FEATURES OF PROGNOSTIC SIGNIFICANCE

	HIGHLY MALIGNANT	MODERATELY MALIGNANT	LOW-GRADE MALIGNANCY
Mitotic figures	present	not found	not found
Multinucleate giant cells	present	rare	not found
Plump nuclei and double nuclei	high incidence	frequent	low incidence
Enchondral bone formation	not found	not found or bizarre and disorganized	frequently present
Prognosis			
a. possibility of cure with radical operation	very poor	fair	good
b. survival uncured	<5 years	±5 years	>5 years

CONCLUSIONS

1. The clinical aspects of cartilaginous tumors of ribs and sternum are reviewed and eleven cases of chondrosarcoma are added to those found in the literature. In the past, poor results have resulted from treatment of cartilaginous tumors, partly because of failure to appreciate the nature of these tumors, and partly from a tendency to surgical conservatism.

2. Cartilaginous tumors of ribs and sternum are either actually malignant or potentially malignant to a high degree. The preoperative differentiation between benign and malignant cartilaginous tumors cannot be made with certainty.

3. The histologic criteria of malignancy in cartilaginous tumors, as established by Jaffe and Lichtenstein, are valid when applied to tumors of ribs and sternum. Prognostic evaluation is possible on the basis of histologic findings provided adequate radical resection is done.

4. Adequate radical resection of chondrosarcomas arising in ribs and sternum entails removal of a long segment of bone of origin, its periosteum, adjacent muscle, underlying pleura, and usually adjacent bony structure.

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CLAMPS FOR END-TO-END ANASTOMOSIS OF BLOOD VESSELS

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SINCE Crafoord performed the first successful operation for a coarctation of the aorta in 1944, several clamps have been devised to make this operation a safer and easier procedure. The clamps devised by Bradshaw and later by Potts have accomplished much in this respect. The instruments described herein have definite advantages and have proved to be very satisfactory in our hands.

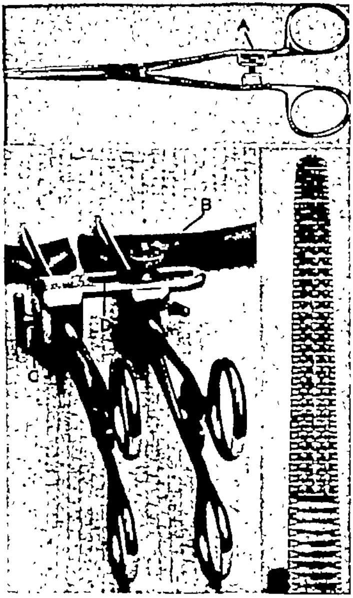

Fig. 1.

These clamps were made by a machinist from two straight Kelly clamps. Two longitudinal grooves were cut over the transverse grooves in the jaws, resulting in three rows of small teeth. The shank of the clamp was bent, as illustrated, so that the ratchet could be replaced by a thumb screw (*A*). The jaws of the clamp were made slightly convex so that equal pressure would be exerted along their entire length when the clamp was applied to the aorta.

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To eliminate the tedious and difficult job of holding the vessel ends in the correct position during the performance of the anastomosis, a small vise was made to hold the clamps in place. The parts of the vise, which were made of brass, may be seen in Fig. 2.

After the vessel has been mobilized, both clamps can be applied and tightened with a thumbscrew (*A*) sufficiently to stop pulsations. The vessel will then remain secure in the clamps and the coarcted area may be resected.

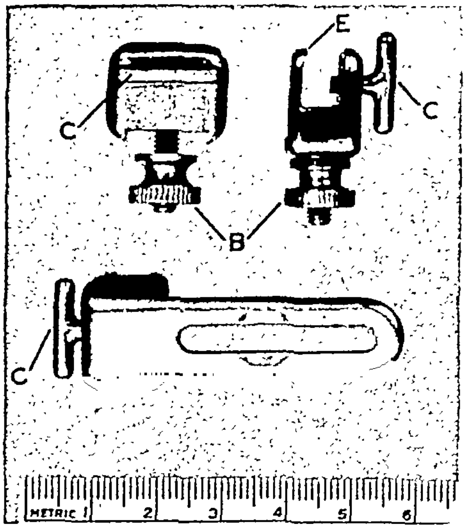

Fig. 2.

The clamps are then placed and locked in the vise by tightening the screw (*C*). A small brim (*E*) prevents the clamp from slipping out.

While the vessel ends are still separated, a stitch may be placed at opposite points of the anastomosis and tied after the clamps are approximated and locked with screw (*B*). The application of the remaining sutures is not difficult when the ends of the vessels are maintained steadily in the proper relationship by the previously described apparatus. The posterior row can be made after the clamps and vise are turned over (as one piece) to the other side.

After the anastomosis is completed, the vise is removed. The distal clamp, followed by the proximal clamp, is slowly released by unscrewing the thumbscrew (*A*).

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Original Communications

PNEUMONECTOMY WITH REPLANTATION OF THE LUNG IN THE DOG FOR PHYSIOLOGIC STUDY

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IT IS well known that many problems of lung and cardiopulmonary physiology remain unsolved. So many complex and variable factors are involved in the responses of this system that our knowledge of the subject often is more theoretical than actual. Previous investigators have learned much from section of the hilum of the lung and perfusion of the isolated organ for short periods of time.[1] By similar methods the cardiopulmonary reflexes have been studied in relation to the problem of pulmonary embolus. Little, however, is known about the entity almost universally called bronchospasm. The concept is useful in explaining many phenomena; actually its existence has yet to be proved. In asthma, for example, it has long been postulated that in certain cases a "trigger point" somewhere in the body can be the initiator of an attack. This theory assumes the existence of reflex arcs which must originate in various sensitive areas. These areas may be in relatively remote parts of the body, such as the skin, pelvic organs, etc. The importance of sensitive areas in the lung itself in initiating reflexes must be further clarifield. Attempts to relieve asthma by interrupting the autonomic nerves to the lung logically follow this unproved hypothesis. The uncertainty surrounding the matter is well illustrated by the opposing views of those advocating the various surgical treatments of so-called intractable asthma.

It seemed to us that a complete denervation of the lung, followed by physiologic studies, would be useful in approaching these problems. We thought that a complete denervation of the lung could best be accomplished by precise dissection of all elements of the hilum, severing these elements completely, then reimplanting the lung by direct anastomosis of all structures.

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TECHNIQUE

The demonstration of the feasibility of the preparation of such an animal is the chief purpose of this report. After several unsuccessful attempts we have succeeded in having a dog survive such an operation. The operation was performed May 26, 1950.* A review of the technical measures involved follows:

Anesthesia.—The method of anesthesia is of prime importance. Preliminary narcosis is achieved with intraperitoneal nembutal. The free communication between the left and right thoraces of the dog demands mechanically controlled alternating pressure anesthesia. Our first attempts were made with the conventional "windshield wiper" type of apparatus, but this proved to be unsatisfactory. Following the report of Dameron and Greene at the April, 1950, meeting of the American Association for Thoracic Surgery,[2] we obtained one of the Burns valve apparatuses, and have found that it greatly facilitates our work, although falling somewhat short of perfection. The machine functions perfectly throughout most of the procedure, delivering an ether-oxygen mixture to an intratracheal tube with an inflatable cuff. It is only during the period of anastomosis of the severed bronchus that supplementary tubing must be introduced into the opposite main bronchus to maintain respiration while the bronchial anastomosis is being done. The operation is a time-consuming one, but is apparently well tolerated with the use of the Burns valve. Intrapericardial 2 per cent procaine is used freely throughout.

Operative technique.—The right chest is opened in the usual manner through the bed of the sixth rib. The azygos vein is clamped, divided, and ligated to facilitate exposure of the hilum. All pulmonary ligamentous structures are then divided, with ligation of minor vessels wherever necessary. The right main bronchus is carefully freed by sharp dissection and meticulous hemostasis back to the bifurcation of the trachea. The bronchus is then divided with a scalpel, and the proximal stump promptly and snugly closed with Allis forceps, allowing respiration to be maintained by the opposite lung. Using the distal bronchial stump for gentle upward traction, the operator proceeds with the dissection of the posterior aspect of the pulmonary artery and veins. In order to obtain sufficient lengths of these structures it is essential that the dissection be carried intrapericardially until (*a*) the trunk of the pulmonary artery is reached, and (*b*) the entrance of the superior and inferior pulmonary veins into the left auricle is demonstrated. Attention is then directed to the anterior aspect of the vessels. The completion of the dissection of the artery offers no further difficulty. In order to visualize the veins fully, however, a plane of cleavage must be developed between the veins and the right auricle. This dissection begins in the pericardial fat alongside the right auricle and proceeds toward the left auricle until the entrance of the veins into that auricle is reached. It should be noted that in the dissection of the veins from the auricle the plane of cleavage must be more or less created, since a natural one does not appear to be well defined. When the dissection is correctly done there should be two or three centimeters each of superior and inferior vein between the auricle and the first branchings. Numerous small bleeding vessels will be encountered and must be controlled. It is also easy to enter the thin-walled auricle inadvertently, a complication greatly increasing the difficulty of the procedure.

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*In a communication received from Dr. Stewart under date of Oct. 26, 1950, it was stated that the animal was alive and in good health. Pulmonary functional studies were being made on it.—Editor.

When the dissection is completed the artery and veins are successively handled in essentially the same manner, the sequence being artery, superior, then inferior pulmonary vein. Each vessel is occluded as near the heart as possible, using a clamp devised by one of us (A. J.), (Fig. 1). This clamp is a modification of the one devised by Freeman[3] for side-to-side vascular anastomosis. It opens at one end so that it may be slipped on and off an intact vessel. It has no long handle, but is controlled by a small thumb screw, thus occupying little space and allowing free access to all sides of the anastomosis.

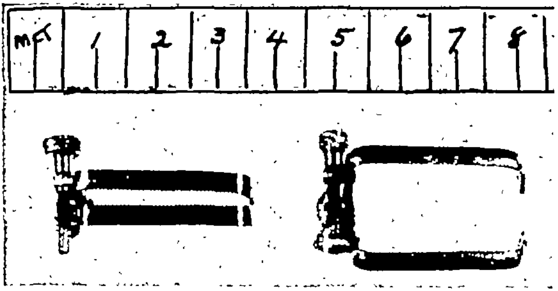

FIG. 1.

Distally the vessel is occluded with a Blalock clamp. After occlusion the vessel is divided and reanastomosis done with 00000 silk by the classical technique of stay sutures converting the round structure to a triangle. Bleeding should be minimal after completion of the anastomoses.

Finally, the bronchus is anastomosed with interrupted sutures of 000 silk.[4,5] It is during this step that the supplementary anesthesia tubing is required. Since the bronchial stump is open, the intratracheal tube cannot be effective and the oxygen-ether under pressure must be introduced through a tube placed in the open bronchus and leading into the opposite main bronchus. As many anastomosing sutures as possible are placed and tied with this tube in place. The remaining sutures are all placed but not tied. The tube is then withdrawn and traction quickly applied to the untied sutures, thereby closing the anastomosis. Anesthesia is immediately resumed through the intratracheal tube. The remaining sutures may then be tied without significant leakage at the line of anastomosis. As much pleuralization of the hilum as possible is then done. There should be prompt re-expansion of the lung following completion of the bronchial anastomosis. The chest is closed in the usual manner. Penicillin is given for five days postoperatively. Anticoagulants have not been used by us.

DISCUSSION

Following survival of this procedure by an animal in our laboratory, various questions must be answered before the preparation can be put to its fullest use as a tool in physiologic experimentation. Our first efforts have been directed toward verification of the mechanical integrity of the reimplanted lung. Before closure of the chest it was directly observed that the respiratory cycle was grossly normal. The lung was well inflated. Pulsations of the pulmonary artery were seen and felt well beyond the line of anastomosis. A free flow of blood was observed in the veins. Thereafter daily percussion and auscultation of the chest, with frequent fluoroscopy, have shown apparently normal ventilation. On the eighteenth postoperative day angiocardiography was done. The pulmonary arterial tree was well visualized with no apparent difference between the right and left sides. These findings led us to believe in the mechanical integrity of the lung.

The functional integrity, in the sense of oxygenation of the circulating erythrocytes, is more difficult to prove. Studies are planned, and have been begun, to determine the oxygen saturation of the blood under varying conditions of: (*a*) unilateral pulmonary ventilation, (*b*) differing oxygen partial pressures in the two lungs, (*c*) contralateral pneumonectomy, and (*d*) contralateral pneumonectomy with reimplantation of the lung. Attempts will be made to induce reflexes by means of chemical and electrical stimulation of the two lungs separately.

These studies will be repeated at regular intervals to follow the effects of nerve regeneration if and when it occurs.

In the near future an attempt will be made to effect an exchange of lungs between two animals.

The severance of the lung lymphatics, as accomplished by this procedure, has caused no observable effects to this time.

CONCLUSIONS

1. A technique is presented whereby a dog's lung is completely denervated by performing a pneumonectomy and replanting the lung by anastomosis of its bronchus, artery, and veins.

2. At the present writing one animal has survived this operation with apparent mechanical integrity of the lung and is undergoing further study to prove functional integrity. The animal is in good health now, five months after the operation. Serial studies are being carried out to investigate regenerative phenomena.

3. It is hoped that the method will offer a useful tool for the further elucidation of problems of pulmonary physiology.

Note.—The authors wish to express their thanks to Doctors Howard Dayman and E. G. Eschner for their advice and aid in the fields of pulmonary physiology and radiography.

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SURGICAL TREATMENT OF THREE CASES OF PLEURAL SARCOMA*

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THE pleura consists of endothelium resting upon a thin basal membrane and a subendothelial layer of connective tissue rich in elastic fibers. Of malignant tumors involving the pleural tissue, the endothelioma is the most common. It probably arises from endothelial cells, and is often called a mesothelioma to indicate the mesothelial character of the cells and their ability to form neoplasms of both epithelial and connective tissue (Maximow and Kux). Malignant degeneration of the subendothelial connective tissue is thought to give rise to pleural sarcomas. Microscopically these growth are often round-celled sarcomas, but also spindle-celled sarcomas have been described, and mixed tumors such as fibro-, angio-, chondro-, and fibroxanthosarcomas are common. While certain forms of sarcoma may be operable, the endothelioma is generally inoperable on account of its infiltrative mode of growth.

Judging by the literature, pleural sarcomas are rare. Seydel (1910) found only one pleural sarcoma in 10,000 autopsies. In 1931 Lichtenstein could trace only thirty-one cases in the entire literature. Since that time no collections have been reported.

Lichtenstein divides sarcoma of the pleura clinically into three groups. The first group is characterized by high malignancy and extensive spread over the surface precluding surgery. Hemorrhagic effusion is regular. In the second group the sarcoma is likewise very malignant but of less extensive spread. It is very infiltrative and often invades the tissue of the lungs; hemorrhagic effusion is common. The third group is much more interesting from a surgical point of view, because many of these cases can be treated radically. They are well demarcated and clinically benign. They are as a rule not attended by pleural effusion. However, their microscopic structure is that of a sarcoma. They differ from the tumors of the first two groups in that they do not metastasize and show no tendency to recur.

Lichtenstein assigns eight of his cases to this third group, but only one of them was operated on (Quincke and Garré). Further descriptions of operated

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From the Surgical Clinic II, Sahlgren's Hospital.
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*It is doubtful that these cases should be called sarcoma. It would seem that Stout's name, "localized, benign mesothelioma" is to be preferred.—Editor.

cases of pleural sarcoma have been published by Jacobaeus and Key (one case), Klemperer and Rabin (one case), and Fawcett (one case).

The majority of the cases of pleural sarcoma reported in the literature were not diagnosed before autopsy. In most of them the tumors were extensive and probably assignable to one of Lichtenstein's first two groups (e.g. Klemperer and Rabin, one case; Jacob, Jais, and Alison, one case; McMahon and Mallory, one case), and in these cases surgery would probably have been of no avail even if they had been diagnosed in an early stage. But also well-demarcated pleural sarcomas have been described (e.g. McNamara, Sargent, and Costich, one case) in which the patients had, however, sought aid so late that their general condition was too poor to permit surgical intervention. Stout and Murray have described a tumor similar to ours which they regard as a localized benign pleural mesothelioma. By using tissue culture methods they found that the tumor actually originated from mesothelium.

Fig. 1.

An important reason why the number of operated cases is so low as compared with the number observed at autopsy is the lack of symptoms of this type of pleural tumor. The disease is not attended by anemia or cachexia. The growth escapes attention until it has attained such proportions as to compress neighboring organs, when the first symptoms such as dyspnea and signs of circulatory insufficiency will appear. But by then the patients are in such a poor condition, and the tumor has extended so much, that radical surgery is impossible.

We have treated three cases of localized pleural sarcoma at our Clinic. All three were discovered incidentally in the course of routine roentgen examination. The patients had been given no trouble by the tumors, all of which had attained the size of a fist.

CASE 1.—405/47. A 35-year-old seaman, who had hitherto felt healthy. When the patient was called up for military service, routine roentgen examination revealed an oval, homogeneous, structureless soft tissue density of 8 by 9 by 9 centimeters (Fig. 1) in the left half of the chest. The tumor, the center of which lay on a level with the bifurcation of the trachea, extended up to the lateral wall of the chest on a level with the fifth rib. It was located interlobularly, and it could not be decided whether it had originated from the lung, the hilus, or the thoracic wall. The aorta could be projected by itself. There was no pleural effusion. Bronchography failed to demonstrate any connection with the bronchi.

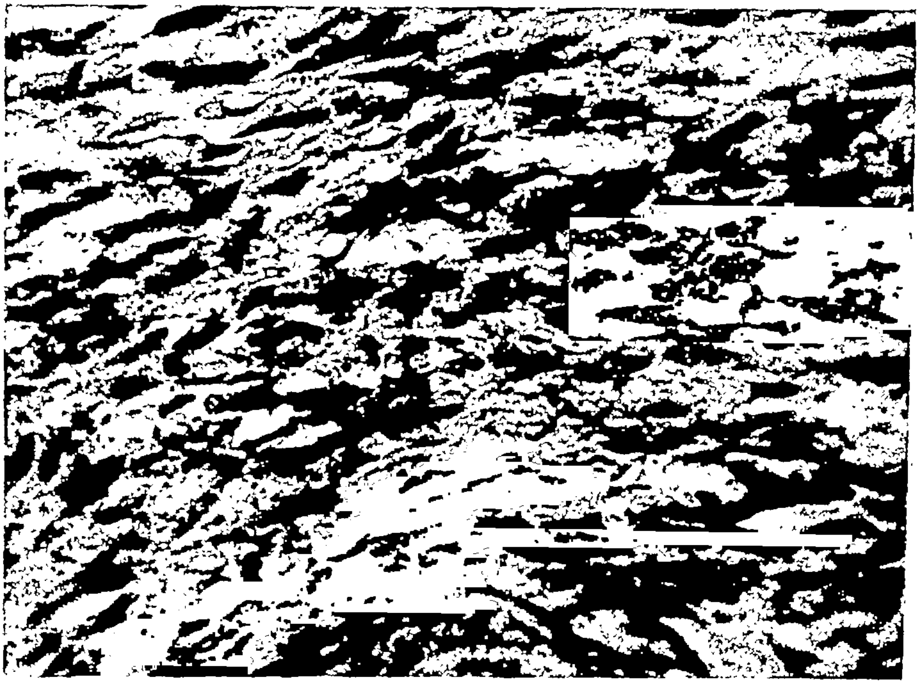

Fig. 2.

Operation.—Wide thoracotomy after removal of the fifth rib was performed. The pleura was then incised under positive pressure, after which the pressure was gradually released, and the lung collapsed. It was observed that the fist-sized tumor had originated from the lateral thoracic wall, to which it adhered with a rather broad base (4 by 4 centimeters) on a level with the fourth intercostal space in the posterior axillary line. The surface of the growth was covered with smooth, shiny pleura, which could be stripped from the tumor without difficulty. At its base it was adherent to the hemiazygos vein, part of which was therefore removed after ligation on either side of the growth. The tumor had apparently grown from the subpleural connective tissue. No connection could be observed with the intercostal nerve or vessels, all of which were dissected and inspected. After enucleation of the tumor the lung was expanded by positive pressure and the wound closed.

The tumor, which was encapsulated, showed a homogeneous cut surface like the flesh of a fish. Microscopically, it showed the characteristics of a fibrosarcoma with a tendency to develop into a spindle-celled sarcoma (Fig. 2). The postoperative course was unevent-

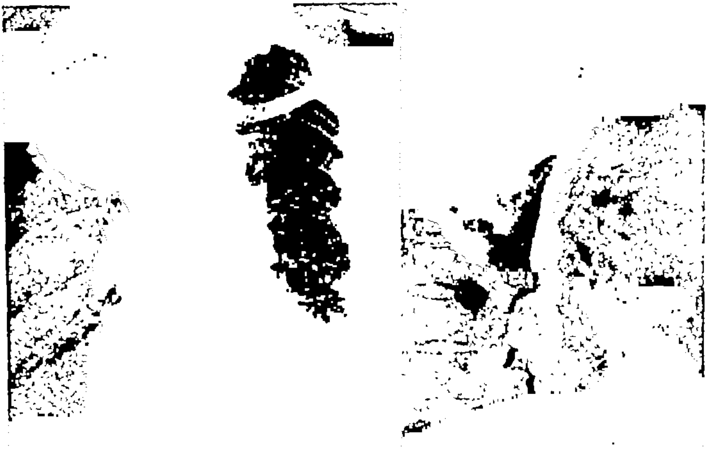

Fig. 3.

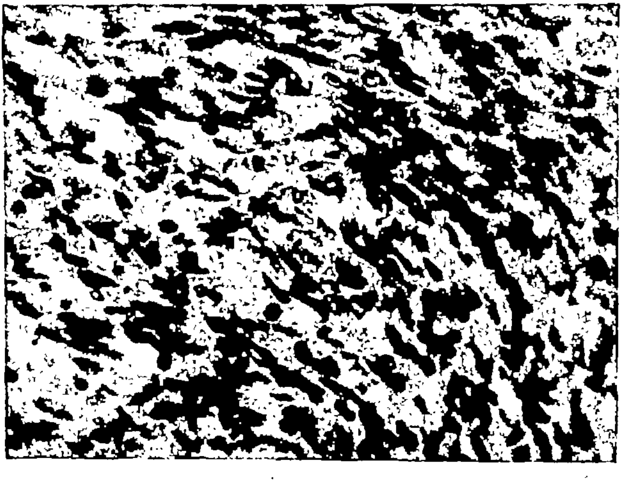

Fig. 4.

ful, except for a moderate pleural effusion, which was aspirated on two occasions. The patient received postoperative radiotherapy. At reexamination two and one-half years after the operation he felt well.

CASE 2.—2609/48. In a hitherto healthy 58-year-old woman who was submitted to roentgenography of the heart and lungs on account of hypertonia discovered by chance, the roentgenogram showed a well-defined, round tumor, about 6 centimeters in diameter, in the right half of the thorax (Fig. 3). The upper border of the tumor was on a level with the fourth rib, while the lower border extended down to the seventh rib. No grooving of the ribs could be observed in the roentgenogram, nor could any fluid be discerned in the pleural cavity. A benign tumor, possibly a dermoid cyst, was suspected.

Operation.—Wide thoracotomy after removal of the sixth rib was performed. After the lung had been collapsed, the fist-sized tumor could be inspected. It was almost round, its surface was smooth, and it had a broad base originating from the fourth and fifth intercostal spaces. The tumor was covered with a smooth, shiny, and somewhat thickened pleura, from which it was easily separated. The intercostal nerves and vessels had not been involved by the tumor, which had not invaded the periosteum of the ribs either. The tumor had presumably arisen from the subpleural connective tissue. Here, as in the previous case, enucleation was easy and apparently radical. The lung was expanded by positive pressure, and the wound closed.

Operation.—Wide thoracotomy by removal of the fifth rib was performed. The fist-sized tumor was seen immediately lateral to the transverse processes and on a level with the fourth rib. The lung was adherent to the entire surface of the tumor from which it was separated by blunt dissection without any damage to the pulmonary tissue. The tumor had a broad attachment (7 by 7 centimeters) to the posterior thoracic wall. The tumor was lined with a somewhat thickened, but smooth and shiny pleura. After a lambdoid incision in the pleura the tumor could be enucleated. Enucleation presented no difficulties, and it was observed that the tumor had originated from the subpleural connective tissue in the fifth intercostal space. The intercostal nerve and vessels were not involved. The pleural defect left after the removal of the tumor was covered with the above-mentioned lambdoid flap. The lung was expanded with positive pressure, and the incision closed.

The tumor was encapsulated, and its cut surface showed a homogeneous, fleshy appearance with a few almond-sized cysts containing a clear fluid (Fig. 6). Microscopically, it showed the characteristics of xanthofibrosarcoma (*Fig. 7*). *The postoperative course* was uneventful, except for a moderate effusion in the pleural cavity, which was aspirated twice. After the wound had healed, radiotherapy was given. At reexamination, three months after operation, the patient was symptom-free.

The three cases described above have much in common. They were all discovered by chance; they were all clinically benign pleural sarcomas (Lichtenstein's third group); they were all fist sized, round, and well demarcated, but with the obvious microscopic structure of sarcomas.

In spite of the proportions they had attained they had given no symptoms, but had all been discovered at routine roentgen examination of the chest, and in middle-aged patients.

In these cases no conclusions can be drawn as to the rate of growth of the tumors. They show, however, that a well-defined density in the roentgenogram ought not to be regarded forthwith as a benign tumor not requiring surgery. We did not resort to exploratory aspiration as a diagnostic aid, because in a few other instances in which the tumor was a dermoid cyst, the cyst content spread outside the tumor, and, if, as in these three cases, the tumors had been malignant, the risks attending exploratory aspiration would probably have been still greater. Neither did we make use of diagnostic pneumothorax, because such a procedure would hardly have influenced the indications for operation. Moreover, we have found pneumothorax to favor serous effusion.

Unless contraindicated for some other reason, all tumors with this x-ray picture indicate surgery, because the tumor might be a sarcoma. In spite of the clinically benign behavior of the sarcomas mentioned above, they grow and thereby compress vital organs, and finally lead to death. In the three cases described above the tumors could be enucleated without difficulty. When carrying out such an operation, however, one must be prepared for lobectomy or pneumonectomy, unless the extrapulmonary location of the tumors can be established at preoperative roentgenography. The tumors respond to radiotherapy, and in the three cases under discussion postoperative radiation was given.

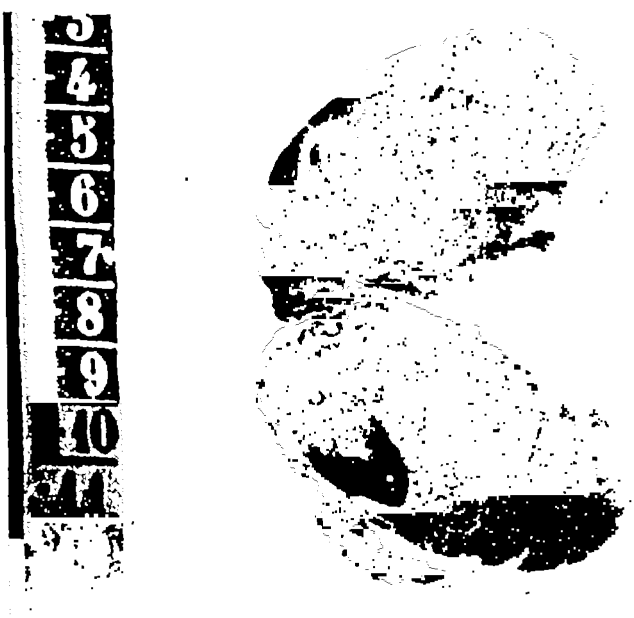

Fig. 6.

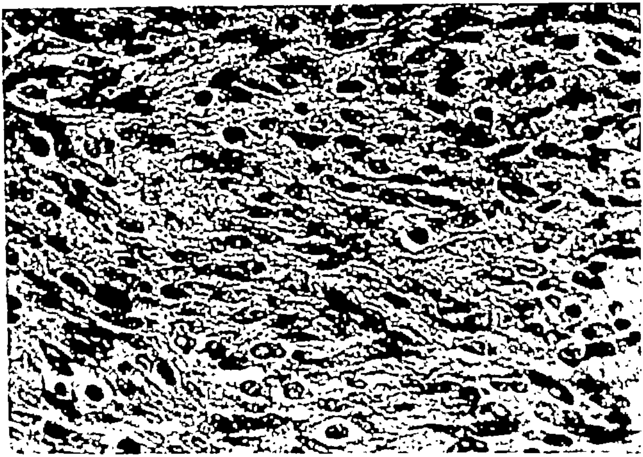

Fig. 7.

SUMMARY

Sarcomas of the pleura are rare. One group of these tumors is of great clinical interest because the growths may be treated successfully by surgery if discovered early enough.

They have the microscopic structure of sarcomas, but do not behave like malignant growths. They are well demarcated, they do not metastasize, nor do they show any tendency to recur. Only some ten cases have apparently been described in the literature, and only a few of these have been operated on.

These sarcomas generally give no symptoms until they have assumed considerable proportions and then compress adjoining vital organs, but by then they are often inoperable on account of the large extent of their growth and the poor condition of the patients.

Three personally operated cases of sarcoma of this type are described. None of them gave symptoms, and all of them were discovered in the course of a routine examination of the chest. They are fist-sized and easy to enucleate.

The importance of surgical treatment of well-defined tumors in the chest is stressed.

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RECONSTRUCTION OF DEFECTS OF THE THORACIC WALL WITH TANTALUM MESH GAUZE

(Preliminary Report of Two Cases)

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THE repair of defects of the thoracic wall has been accomplished in various ways, as outlined below. But, we believe that these are the first reported cases in which tantalum mesh gauze has been used. When tumors of the chest wall are radically removed or trauma leaves defects, herniation of the lung may occur unless some firm reconstruction is performed.

Tuffier,[13] in 1891, treated a lung hernia of the second intercostal space by removing the sac and closing the pleura. He gives no definite description of any repair of the outer layers. Volger,[14] in 1898, presented a case of lung hernia near the right border of the sternum, following fracture and displacement of the right third rib. He suggested that an osteoperiosteal flap of the sternum or second or fourth ribs could be made and swung into the defect. Vulpius,[15] in 1900, operated on Volger's patient, and split the second rib and costal cartilage near the sternum into an anterior and posterior half of bone and cartilage. Then he swung the medial end of the anterior split portion down, and attached it to the sternal remnant of the third rib with silver wire. The lateral end remained attached in its normal position. The hernia recurred after one year. Graham,[5] in 1922, treated a cervical lung hernia by packing the region with iodoform gauze and removing the gauze five days later. Montgomery and Lutz,[9] in 1925, repaired a hernia of the fourth anterior interspace by turning a flap of periosteum and perichondrium from the anterior surface of the fifth rib and cartilage into the fourth interspace. An osteocartilagenous flap of the fourth rib was then made by splitting it in two from above downward, and turning it down, hinging it on its periosteum and perichondrium. Goodman,[4] in 1933, repaired a hernia of the fifth interspace by dissecting out the pleural sac, and closing it after the redundant pleura had been removed. Then the anterior periosteum was turned down from the fifth rib and up from the sixth rib, and sutured over the weakness. Winkel,[17] in 1935, wove strips of fascia lata between the ribs or endothoracic fascia to close a weakness in the thoracic wall. Watson and James[16] modified this technique by closing the defects with single, large, free fascia lata grafts. Maurer and Blades,[8] in 1946, used periosteal flaps and ribs cleaned of periosteum, cut across, and swung obliquely down to cover defects. They reported that Paulson had attempted to use a tantalum plate to cover a hernia of the lung, but had abondoned it because of the difficulty of immoblization. Beardsly[1] used tantalum plate in three cases

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when reconstructing the chest wall after resecting large areas. The patients were comfortable, but in the three cases they developed serous drainage from around the plates, and in two cases the serum became purulent. The plates were later removed in all three cases, after there had been a fibrous reaction around them giving stability to the thoracic walls. Campbell[2] reconstructed the entire thickness of the anterior thoracic wall by freeing the latissimus dorsi muscle from its insertion and swinging it up into the defect, and covering the muscle with a free split thickness skin graft. When the skin is involved in the tumor and has to be sacrificed, this method has advantages over the others that have been described. The surface can be epithelialized by a free graft, and dependence is not placed on flaps of skin and subcutaneous tissue, whose survival may be precarious.

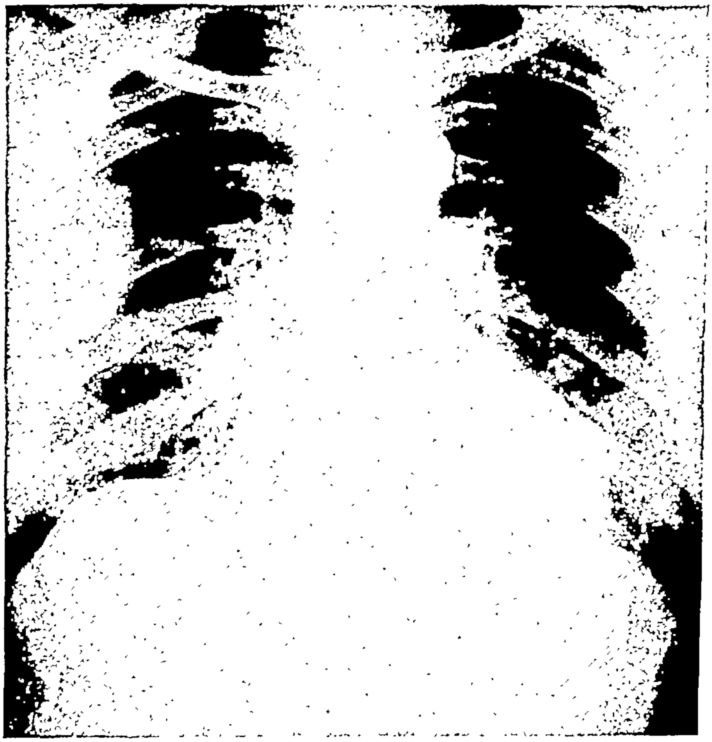

Fig. 1.—Preoperative roentgenogram of the chest in Case 1, showing tumor of the right chest wall with partial destruction of the sixth rib.

When the skin and subcutaneous tissue do not have to be removed, we have used tantalum mesh gauze in reconstructing the thoracic wall. Tantalum mesh gauze is more pliable than plate, and has been used successfully in the repair of a variety of hernias, ventral, indirect inguinal, direct inguinal, obturator, and recurrent inguinal.[6, 12] More conservative treatment consisting of a corset with a pad or obturator over the deep defect can be used, but it is not curative.[10, 11] One prefers not to bury a foreign substance when repairing a thoracic hernia, but if the defect is large this method may offer a satisfactory solution. In the two cases presented herein, tantalum mesh gauze (50 by 50 mesh of .003 inch tantalum wire) was used to reconstruct large thoracic defects.

CASE REPORTS

CASE 1.—St. Luke's Hospital, No. A 160-202. A 51-year-old white man was admitted on Dec. 18, 1948, with intermittent pain in the right infrascapular region, usually brought on by coughing. He had a chest roentgenogram taken seven months before admission which was negative. When the pain persisted cholecystogram followed by a roentgenographic examination of the colon were performed, and the only abnormal finding was diverticula of the colon. There was no history of hemoptysis, productive cough, or weight loss. One week before admission, or approximately seven months after the first roentgenogram, a second roentgenogram of the chest revealed a tumor of the lateral chest wall extending from the level of the second to the fourth anterior interspaces. The tumor had apparently destroyed the sixth rib in the region of the axilla. The main density projected medially for approximately 5 centimeters (Fig. 1).

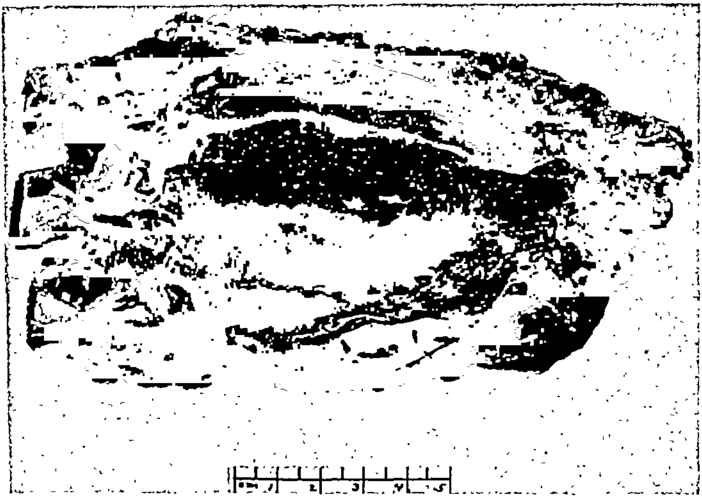

Fig. 2.—Tissue including the tumor, removed from the right chest wall in Case 1. Photograph of the pleural surface.

On physical examination the patient was a well-developed man who did not appear acutely or chronically ill. Temperature was 98.8° F., blood pressure 126/74. Eyes, ears, nose, and throat were normal. Lungs were clear to percussion and auscultation, and no masses could be felt in the thoracic wall. The liver was at the costal margin. No abdominal masses were felt. Neurologic examination gave essentially normal findings.

Laboratory examination showed a hemoglobin of 60 per cent (8.8 Gm.), red blood cells 3,600,000, white blood cells 5,300, polymorphonuclear leucocytes 60 per cent, lymphocytes 36 per cent, monocytes 4 per cent. There was moderate hypochromia and slight anisocytosis. Urinalysis showed an acid reaction, a specific gravity of 1.020, a 1 plus albumin, and was negative for Bence-Jones protein. On microscopic examination an occasional white blood cell was seen, and there were numerous hyaline casts. Urea nitrogen was 11.8 mg. per cent. Serum proteins were 6.8 Gm. per cent, albumin 4.0 Gm. (60 per cent), and globulin 2.8 Gm.

citrated whole blood on his third postoperative day; on the same day he developed auricular fibrillation, and signs of fluid in the right thorax. The auricular fibrillation subsided spontaneously, and right thoracentesis yielded 300 c.c. of bloody fluid. A right thoracentesis was done again on the fifth postoperative day, and 275 c.c. of bloody fluid were removed; 80 c.c. and 50 c.c. of serous fluid were aspirated from the wound on the fifteenth and seventeenth postoperative days. No further thoracentesis or aspiration was required. He was discharged on his twenty-first postoperative day without sedatives or analygesics.

The patient has been given urethane treatment by Dr. N. L. Higinbotham of Memorial Hospital, New York City; and at this time, one year after operation, there is no suggestion of other bone lesions. The patient's right thorax is comfortable, and there is no lung hernia. Upon deep inspiration there is slight decrease in expansion on the right side. Follow-up chest roentgenograms, eight months after operation, are shown in Figs. 4 and 5.

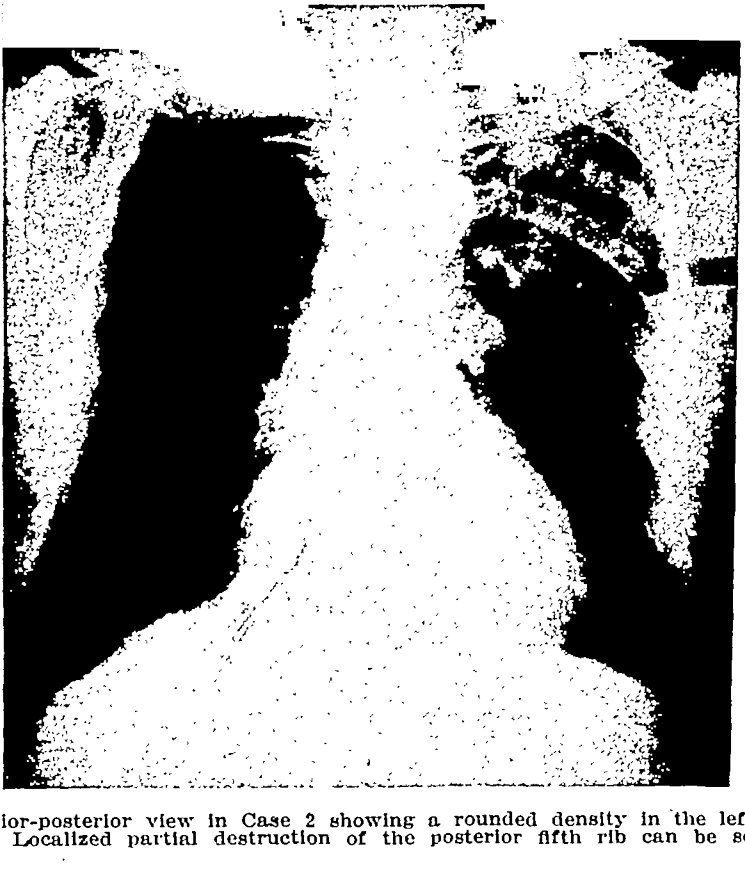

Fig. 6.—Anterior-posterior view in Case 2 showing a rounded density in the left upper lung field. Localized partial destruction of the posterior fifth rib can be seen.

Case 2.—St. Luke's Hospital, No. 178-019. A 50-year-old man was admitted on Feb. 1, 1950, with precordial pain of five days' duration, which began while bending over. He had two coronary occlusions in 1945 and a third in 1947, for which he had been admitted to another hospital. After the onset of pain, a physical examination and an electrocardiogram were done at the hospital where he had previously been treated for coronary artery occlusions. No changes were noted, and the patient was sent home. The pain became more and more severe, and began to radiate down the left arm. Coughing and deep breathing were painful, and there was expectoration of thick yellow sputum.

Moderate dyspnea and orthopnea had been present since 1945, but there was no history of ankle edema, weight loss, chills, fever, hemoptysis, or night sweats.

On examination the patient appeared older than his stated age. Temperature was 98.4° F., blood pressure 122/74, pulse 80, and respirations 22 per minute. Eyes, ears, nose, and throat were normal. The chest showed an increased anterior-posterior diameter and fair expansion, and was clear to percussion and auscultation. There was deep tenderness elicited by percussion in an area 3 cm. by 3 cm. just to the left of the left nipple. The heart sounds were of fair quality and regular, without murmurs or thrills. On examining the abdomen, the liver descended 2 cm. below the costal margin on deep inspiration and was quite tender. No abdominal masses were felt. Rectal examination was negative. The prostate gland was of normal size and consistency. Proctoscopy to eight inches was negative except for small internal hemorrhoids.

Laboratory examination showed a normal red cell and white blood cell count, and urinalysis. Blood Mazzini was negative, blood urea nitrogen 12.1 mg. per cent, blood sugar 79 mg. per cent, serum phosphorous 2.8 mg. per cent, serum alkaline phosphatase 4.1 Bodansky units, serum acid phosphatase 3.3 Gutman units, erythrocyte sedimentation rate 26 mm./hr. The sputum was negative for acid fast bacilli. The stools were negative for occult blood.

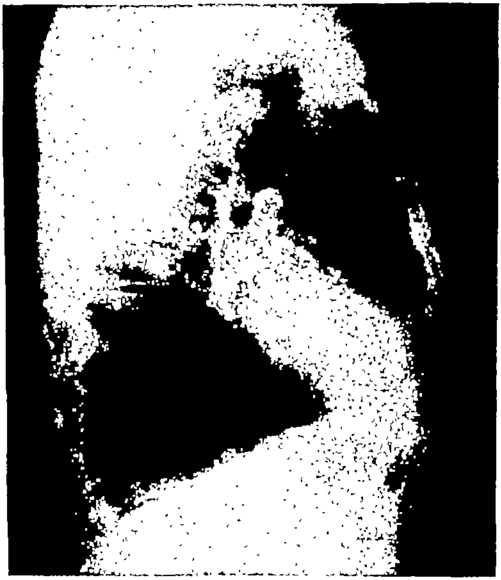

Fig. 7.—Lateral view in Case 2 showing the density situated in the upper posterior thorax.

An x-ray examination of the chest showed a tumor, 10 cm. in diameter, situated in the posterior portion of the left upper lung field. There were increased vascular markings, obliteration of the left costophrenic sinus, and slight cardiac enlargement. Repeated roentgenograms showed progressive increase in the size of the lesion, and localized destruction of the fifth rib (Figs. 6 and 7). Bronchoscopy was negative, and bronchial washings examined by the Papanicolaou technique were negative for malignant cells. Skeletal x-ray survey failed to reveal any other bone lesions. Gastrointestinal and urological investigations showed no abnormalities. Electrocardiograms gave evidence of an old posterior coronary occlusion.

Six weeks after admission a left thoracotomy was performed under endotracheal nitrous, cyclopropane, and ether anesthesia. The thorax was entered through the sixth interspace, and a tumor approximately 10 cm. by 10 cm. by 4 cm. was seen attached to the fourth, fifth, and sixth ribs just to the left of the transverse processes. It was also attached to the periphery of the left upper lobe. A block excision of approximately 15 cm. square of the chest wall was performed consisting of fourth, fifth, and sixth ribs, intercostal muscles, parietal pleura, tumor, and peripheral portion of left upper lobe. A 15 square centimeter piece of tantalum mesh gauze was then made with the edges doubled under, to fit the defect. Drill holes were placed in the adjacent ribs, and interrupted tantalum wire was used to hold the mesh in position. A Foley bag catheter was placed through a stab wound in the eighth interspace and attached to underwater drainage. The outer structures of the chest wall were closed as in the previous case. During the operation the patient received a transfusion of 500 c.c. of citrated whole blood.

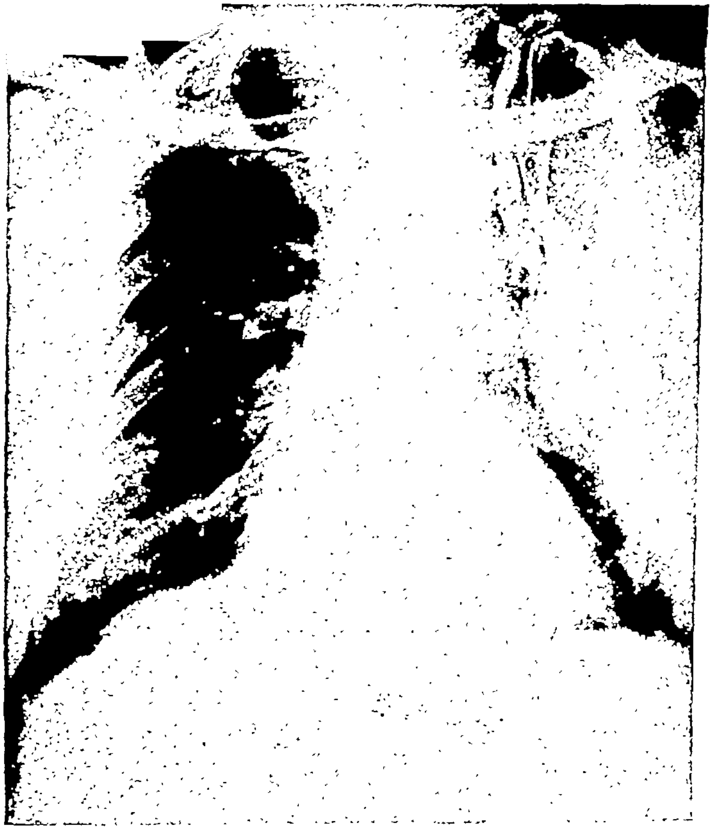

Fig. 8.—Anterior-posterior view in Case 2, one month postoperatively.

On gross pathologic examination the specimen consisted of a tumor mass 7.5 cm. by 8 cm. by 4 cm., attached to the inner surface of three ribs, each of which was 13 cm. in length. The inner surface of the tumor was covered with smooth, shiny serosa except for a ragged section of lung tissue. On microscopic examination there were sheets and cords of tumor cells tending to form small glands. The cells varied in size and shape, and in one section were invading bone marrow. Diagnosis was metastatic adenocarcinoma involving the chest wall. There was nothing in the histology to suggest the origin of the tumor.

Postoperatively, the patient received 200,000 units of penicillin intramuscularly every six hours for the first eight postoperative days. The temperature rose to 102.2° F. on the first postoperative day, and fell to normal on the second postoperative day, where it remained. The catheter was removed from the thorax on the seventh postoperative day, and no aspirations of the thorax were required. There was pain in the incision following operation, but it gradually decreased, and the patient was comfortable at the time of discharge on the twenty-first postoperative day.

On physical examination one month after operation the incision was well healed, with only slight tenderness in the region of the repair, and there was no evidence of lung hernia. There was slight decreased expansion on the left side when taking a deep breath. Check-up roentgenograms showed the tantalum mesh gauze to be in position in the defect; most of the tantalum wire sutures, however, were broken (Fig. 8).

DISCUSSION

In the two cases presented, tantalum mesh gauze has been well tolerated by the tissues during the relatively short period of observation. We believe it is superior to tantalum plate, when the defect occurs in the region of the ribs, where there is motion and shearing stresses and strains. The mesh can be fashioned to fit any type of defect, and is readily fixed in position with tantalum wire to bone or soft tissue. The plate would probably be more useful for repairing defects of the sternum, or for stabilizing the upper anterior thorax after removal of the manubrium and sternal ends of the clavicles. The gauze has remained in situ, and the patients have been fairly comfortable since the time of discharge. The tenderness displayed in the region of the repair of the second patient may be due to the irritation of the broken wires.

The tumor in the first case was to all appearances a solitary plasma cell myeloma. In such cases, destruction or removal of this type tumor appears justified.[3, 7] In the second case, the tumor was an adenocarcinoma of as yet undetermined origin. Operation has afforded the patient relief from pain, but there is some tenderness in the region of the repair.

SUMMARY

1. Methods of reconstruction of the thoracic wall have been reviewed.

2. A method of repair of the thoracic wall is presented, wherein tantalum mesh gauze has been used to fill the defect of the bony thoracic wall. Two cases with this type of reconstruction are presented.

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OBSERVATIONS ON DIVISION OF ADHESIONS IN OPAQUE LOBES

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IN THESE days, when the treatment of pulmonary tuberculosis seems to oscillate between such extremes as streptomycin at one end and lung resection at the other, both being recent acquisitions in the treatment of this disease, to talk of "black-out" lobes in artificial pneumothorax in relation to healing may sound rather out of place to some. This subject has been investigated and discussed at great length by several authors years ago, and although opinions are still divided about its beneficial or dangerous effects, I feel that the last word has not yet been said, and further contributions to its clarification should still be welcome.

From time to time reports have appeared in the Journals devoted to tuberculosis where, after having operated on a few hundred cases of artificial pneumothorax, the author feels obliged to tell us about his results with the Jacobaeus operation.

These results are usually given with a great profusion of figures, charts, and tables. The conclusions almost invariably contain figures on sputum conversion, cavity closures, pleural complications, cures, improvements, and so on, thus putting down into arithmetic something so variable and poliomorphic in its very nature as is the behavior of tuberculous lesions in different individuals.

From the review of such papers one principle seems to stand out and dominate the whole field of this operation, namely the mechanical conception that a successful Jacobaeus operation is one in which all adhesions have been severed, wherever they are, whatever their type may be, regardless of their number. Fortunately, all the authors are agreed on the dangerous practice of leaving broad adhesions halfway cut, particularly if they are apical and close to the tuberculous lesions.

I often wonder how much scientific ground there is for the widespread belief that adhesions are the main source of pleural complications in the pneumothorax; also for the other accepted principle that a pneumothorax with adhesions cannot be a good pneumothorax. In pulmonary tuberculosis nothing can be easier than to prove anything, from the senseless contention that early hemoptysis can be a sign of good prognostic significance to the not so senseless theory that pleural thickening may encourage healing by starting a process of peripheral fibrogenesis.

Side by side with an ineffective pneumothorax free from adhesions it is quite easy to put another one, where, despite "inseparable" adhesions there

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exists selective collapse, and yet the result has been satisfactory. This is not only so in relation to sputum conversion, but also where closure of cavities is concerned. The nature of tuberculous lesions and individual resistance to the disease is such that the closure of cavities and resolution of infiltrative lesions does not always come about by such a simple maneuver as the cutting of a few bridges of pleural tissue. The *importance of pleural adhesions in pneumothorax* is not a quantitative factor, but rather one of quality. More important than the adhesion itself is its relation to the underlying lesion, its influence, unfavorable or even favorable toward the progression or regression of the diseased area. Everybody has witnessed the progression of a tuberculous lesion despite a good pneumothorax, and vice versa.

The tendency nowadays, however, is to submit more cases of pneumothorax for early thoracoscopy with a view to division of adhesions, even if none can be seen in the roentgenogram.

I feel that the importance of the presence of adhesions in pneumothorax has been rather overestimated, probably because it is a relief to our conscience to feel that should a complication appear in the course of refills, no blame can now be pinned on such an elementary precaution as is the severing of any offending adhesions that may be present. As if a pneumothorax with or without adhesions were not, in itself, a dangerous procedure! "Walking on the edge of a volcano" Robert Coope has called it.

Needless to say, the excellent results obtained in well-selected and skillfully managed cases, justifies the increasing use of this form of treatment, and this being so, we must be prepared, now and then, to meet such complications as pleural effusions, spontaneous pneumothoraces, etc.

Alexander and Deist estimated that only 14, or 25 per cent of all cases of pneumothorax are suitable for adhesiotomy. About 40 per cent of pneumothoraces are unsatisfactory on account of the presence of offending adhesions (Matson). From the results obtained by many authors, 60 per cent of these pneumothoraces can be converted into satisfactory ones by division of the adhesions, otherwise only 15 per cent of them may prove to be worth while continuing with.

As for complications, I think it is safe to put the incidence of pleural effusions, large and small, at 40 per cent of all cases submitted to adhesiotomy, 8 per cent of them turning empyematous soon, or later on after the operation if the treatment has been continued for longer than six months.

With regard to these complications I feel it is only fair to say that they do not represent a regular event in the routine work of a chest surgeon. I feel that the highest incidence of empyemas following adhesion section in any given series represents the unpleasant tribute paid by the surgeon for his first hundred cases. The incidence of pleural complications decreases gradually proportionate to the skill and experience of the surgeon. I wonder how many of the 19 per cent of empyema cases of Matson, 14 per cent of Matill-Jennings, 8 per cent of

Goorwitch, and 13 per cent of P. W. Edwards did happen among the first hundred cases operated upon, and if they would get the same figures today with their broader and much increased knowledge of the technicalities of this operation and better selection of cases, etc.

On reviewing the work on the Jacobaeus operation, published by Jacobaeus himself, Alexander, Anderson, Matson, Brock, P. W. Edwards, Cutler, Marcus Pinner, Xalabarder, Laird, Aubrey-Jones, Goorwitch, Stemmerman, Weber, Herholtz, O'Shaughnessy, Newton, Chien, and others, I find that special emphasis has been placed on such items as sputum conversion and caivty closure, undoubtedly the main objectives of this operation. Next, these authors concentrate their investigation on operative and postoperative complications, technique employed, results according to the extension of the disease, etc., but no information can be found as to the radiologic and pathologic types of the disease, a point of considerable importance. Apart from Coryllos who left us an enthusiastic defense of the beneficial role played by atelectasis in pneumothorax and other papers dealing with this subject along polemical lines, no practical work can be found on a large series of such cases. It is not reasonable to be dogmatic either for or against lobar atelectasis in pneumothorax just because bad or good results have been obtained in a small group of cases. "Black-out" lobes may result in cavity closure and sputum conversion in some cases, and may also bring about a dangerous state of affairs for reasons not yet well established. This paper is an attempt to bring more light on the interpretation of this apparent paradox, and is based on the experience of over 300 cases of the Jacobaeus operation. A total of 57 cases of opaque lobes have been studied and compared. As a result it is felt that the beneficial effects of lobar atelectasis observed in one group of cases and the bad results obtained in another are not solely due to the classical division of tuberculous lesions into productive and exudative—other factors also play an important role. Cases of transient or temporary atelectasis observed during pneumothorax are not considered in this series.

Of the three main theories put forward for the explanation of "opaque lobes" in pneumothorax, namely, bronchial obstruction (Coryllos), pneumonitis (Pinner), and active contraction of the alveoli (Xalabarder), we feel that none can be accepted as a single causative factor. By Bronchography it has been shown that in a number of cases of opaque lobes lipiodol may not only fill the main bronchi and secondary branches, but also the most distal alveolar territories, and this is taken to be a clear proof that there can be lobar atelectasis without bronchial occlusion or exudative engorgement.

By thoracoscopy it is quite common to see small pink patches of aerated lung tissue amid large areas of airlessness—"breathing zones" of Xalabarder. Radiology and thoracoscopy have shown that there are two main types of opaque lobes in pneumothorax: the flabby, wrinkled, bluish-colored lobe, triangular in shape; and the tense, roundish, shiny, grayish-colored, densely opaque one. This distinction in color, consistency, and shape is paramount because the prognosis of the pneumothorax in one case or the other is vastly different.

As a rule, on doing a thoracoscopy with a view to adhesion section, the first movement of the surgeon is to find out whether there are any adhesions present, the type of these adhesions, where they are inserted, if they are operable or not, etc., and little or no attention is paid to the appearances of the collapsed lung. The observation that cavity closure in opaque lobes is not necessarily the consequence of complete adhesion section led me to follow closely the behavior of this type of pneumothorax ever since 1943. In my experience total, incomplete, or partial division of adhesions in roundish and solid opaque lobes with cavities within does not alter matters much. If incompletely freed the cavity persists in the middle of the homogeneous density, or even may "balloon." If freed completely it simply descends and gravitates over the remaining aerated lung by its heavier weight, giving quite often rise to atelectasis of the whole lung, a dangerous state of affairs and one which would call for hasty reexpansion measures such as withdrawal of air, frequent change of posture, even bronchoscopic suction, this last procedure of no avail in sudden reflex apneumatosis.

On the contrary, triangular-shaped opaque lobes give a great percentage of cavity closures even without adhesion section, provided that these adhesions are not inserted against mobile structures of the chest wall. I am not advocating the discontinuance of the practice of adhesiotomy in these cases. As many adhesions as possible should be severed in any case of pneumothorax, but inoperability or incomplete division is not always synonymous with failure. Alexander and Anderson found that 41 per cent of their cases had incomplete division of adhesions, yet in 28 per cent the result of the treatment was satisfactory, and in 16 per cent definitely improved. Goorwitch had 69 per cent of

TABLE I

TYPE OF OPAQUE LOBE	WITH DIVISION OF ADHESIONS						WITHOUT ADHESION SECTION		TOTAL	
	GOOD RESULTS			BAD RESULTS			GOOD	BAD	GOOD	BAD
	ALL ADHESIONS CUT	SOME ADHESIONS LEFT	MAIN ADHESION LEFT HALF-WAY CUT	ALL ADHESIONS CUT	SOME ADHESIONS LEFT	MAIN ADHESION LEFT HALF-WAY CUT				
Triangular "Opaque Lobe" (Either before or after operation)	2	11	4	1	1	3	3	-	20	5
Round "Opaque Lobe" (Either before or after operation)	4	-	-	5	1	10	1	-	5	16
Whole Lung "Opaque"	1	-	1	1	2	2	2	2	4	7

incomplete divisions, yet 44 of 102 cases of incomplete adhesiotomy showed satisfactory relaxation of the diseased lung. Aubrey-Jones had 52 incomplete divisions of 401 cases, with 13 per cent of the former successful enough to justify their continuance. In Matson's series of 206 cases, 83 could not be completely divided, and 16 of these cases (7.7 per cent) did quite well. Unverricht had 26 per cent of good results in the incomplete division group, and Gravesen's figure for the same group was 33 per cent.

Unfortunately, for the support of my thesis, none of these authors specify the type of lesions present in those percentages of good results following incomplete adhesiotomy. Also, the term "incomplete" may mean half-way-cut adhesions or partial division only (some adhesions being left undivided).

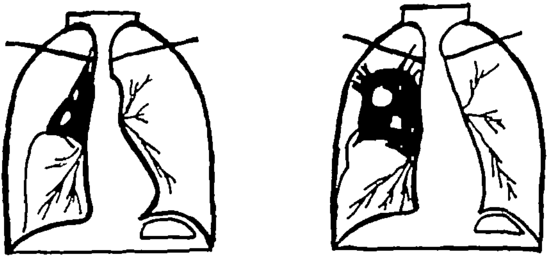


Fig. 1.—Triangular shaped "opaque lobe" (beneficial). Roundish "opaque lobe" (dangerous).

In the series presented here on the results of the Jacobaeus operation in opaque lobes, the best results have been obtained in the "partial division of adhesion" group, where one or two adhesions had to be left undivided and attached to the upper mediastinum or to an immobile structure of the chest wall. This apical attachment appears to act as an anchorage which would stop the apex from falling downwards, also contributing to the triangular shape of the lobe. If this apical anchorage is considered to be harmful on account of its tensive action on the underlying lesions and there are *also* diaphragmatic adhesions, thus making this tension a longitudinal one, the rational thing to do next is to paralyze the corresponding hemidiaphragm, resorting even to a short period of pneumoperitoneum (see Table I), if necessary, before abandoning the pneumothorax.

Once established, this distinction between triangular and roundish opaque lobes, the former beneficial, the latter dangerous, it is essential that we should try to find out the reasons for their different behavior. Why is it that in one case the lobe would collapse almost immediately, after a few refills, giving the impression of being soft, wrinkled, with a tendency to gradual and progressive retraction, while in the other cases the opaque lobe remains always solid, tense, really opaque, maintaining its consistency and spheroidal shape with or without adhesion section. Also, why is it that in the first group cavities tend to close readily, while in the second group they seldom do, if at all? The answer to these questions must surely lie in the underlying anatomopathology of the affected lobe prior to the pneumothorax (Fig. 1).

With this in mind I have reviewed and compared the films of 57 cases of opaque lobes, before and soon after induction of the pneumothorax. They fall into two main groups, one termed "preatelectatic stage" by Xalabarder or "spastic lung" by Tapia, in which the affected lobe appears decreased in size, the mediastinum displaced toward the diseased lung, and the corresponding hemidiaphragm raised. The lobe is not completely "cut off" from the pulmonary aeration as can be proved by bronchography (Figs. 2*A* and *B*). The pulmonary pattern has not entirely disappeared, and, although there may be triangular or fanlike areas of shadow radiating from the hilum (usually the posterolateral segment), the apical and pectoral segments may still remain aerated. It is quite possible that there may be a certain degree of bronchial dilatation, but this bronchiectasis is rather a "dry" one, uncomplicated, harmless. Not infrequently there is a system of two or three small cavities within the lobe. By bronchoscopy the opening of the upper lobe appears quite patent and healthy. Clinically there are no physical signs of bronchial stenosis whatever; there is little cough and little sputum, no systemic disturbance, and the general condition of the patient quite disproportionate to the extension of the

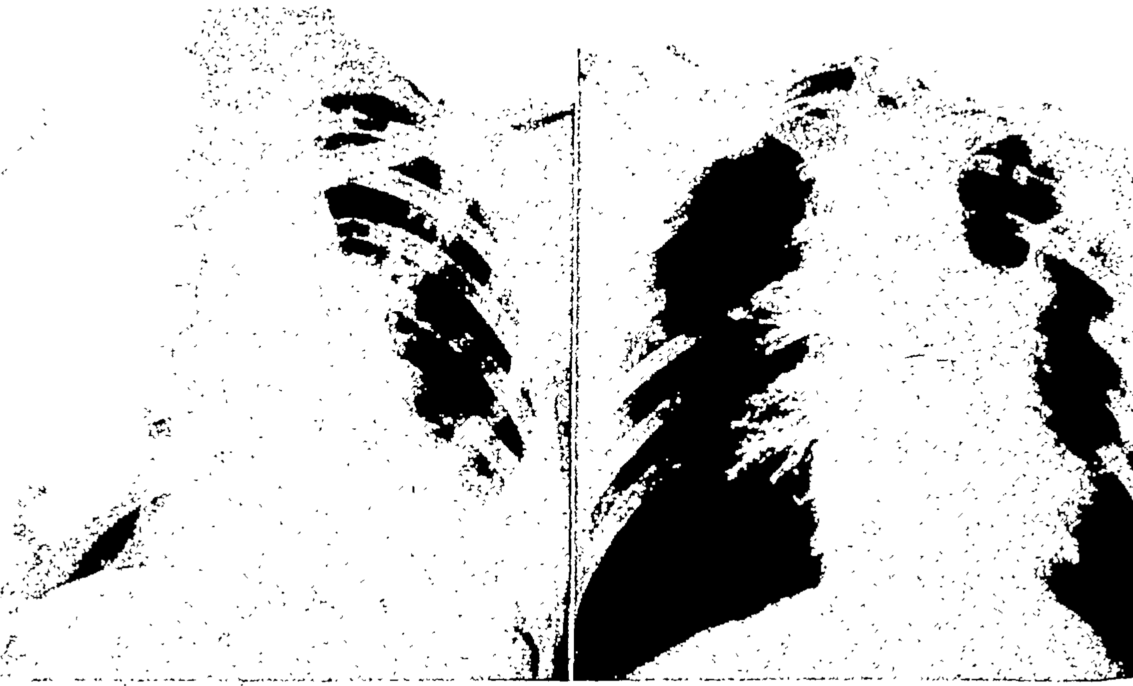

A. *B.*

Fig. 2.—*A*, case of beneficial "opaque lobe" resulting in cavity closure. Bronchography has failed to show blockage of the main divisional branches of the upper lobe. (Fig. 2*B*.)

pulmonary lesions. The study of this group of lesions rather recalls some of the cases described under different terms by Ornstein and Ulmar, Davidson and Ellman.

It appears that histologically the dominant feature is the disappearance of *the alveolar pattern and the reduction of the blood flow.* There is marked *interstitial fibrosis, hialinization and collagenous changes around the bronchi and* blood vessels. *This results in distorted and narrowed* bronchioles where oxygenation of the blood must be negligible. The alveolar walls are thickened by the luxurious fibrogenesis and concomitant emphysema present. Reversibility of these changes is a very remote possibility, and is much hindered by the

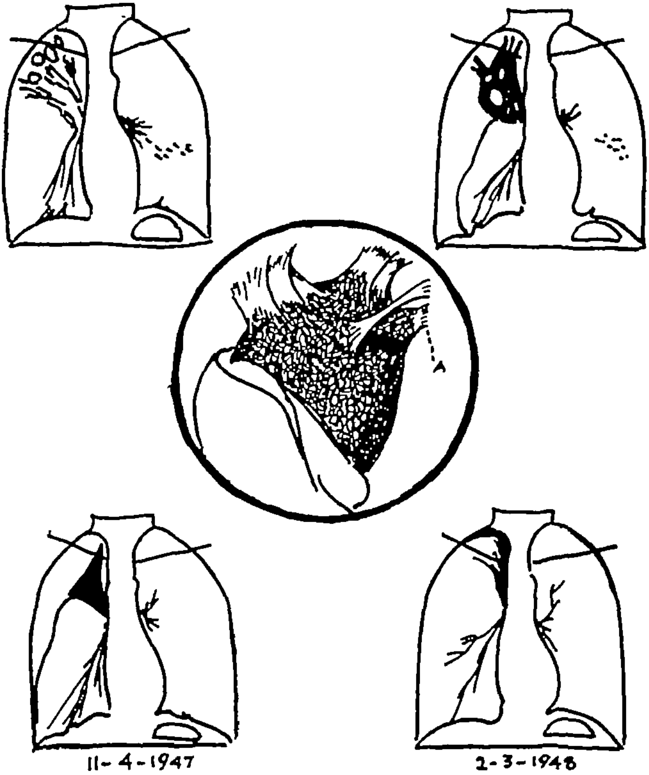


Fig. 3.—Case of beneficial "opaque lobe." Drawings reproduce radiological appearances before pneumothorax, after induction, thoracoscopic view (adhesion *A* left undivided) after adhesion section and final result.

fibrotic nature of the whole process. In a way this type of benign "lobitis" is to pulmonary tuberculosis what chronic interstitial pneumonitis is to other acute pneumonic and bronchopneumonic conditions of the lungs (Figs. 3, 4, and 5).

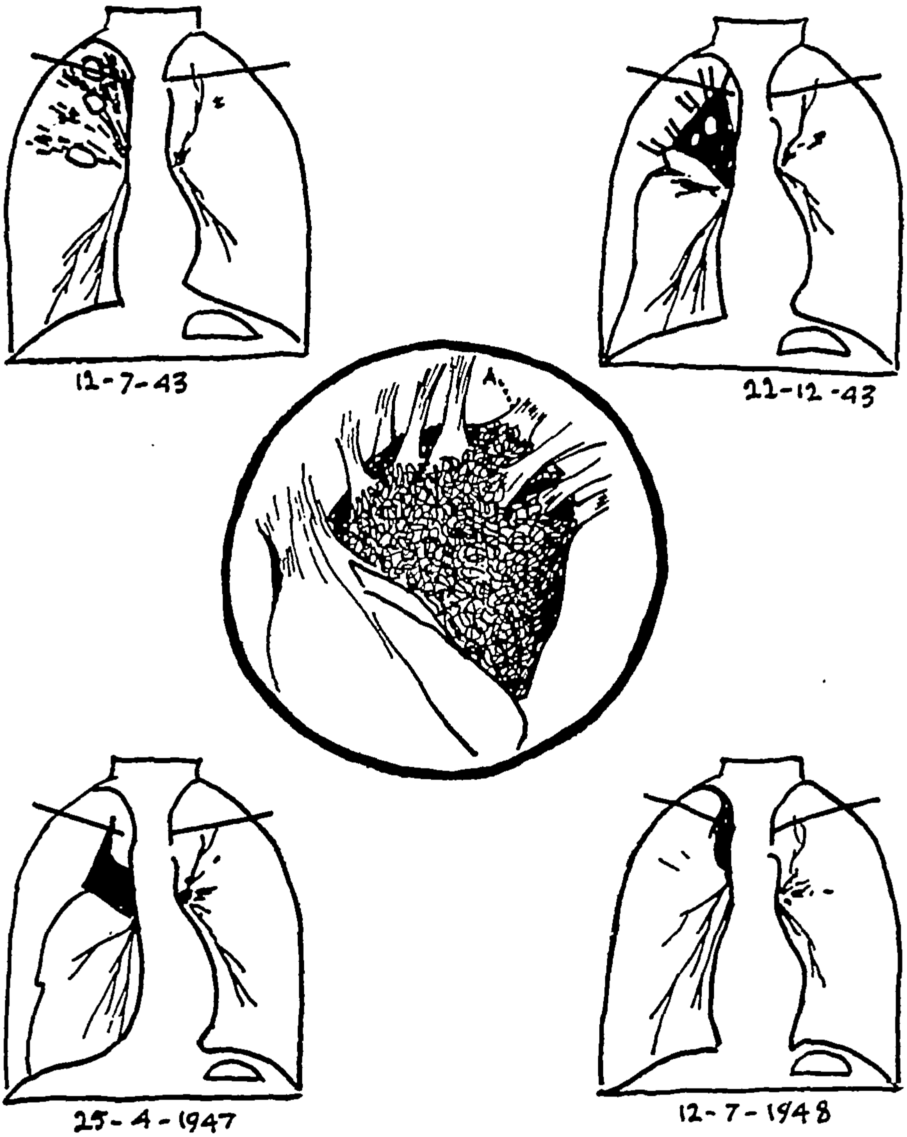


Fig. 4.—Case of beneficial "opaque lobe." Drawings reproduce appearances before induction of pneumothorax, soon after, thoracoscopic view (adhesion *A* left undivided), appearances following adhesion section and final result.

In the other group the roentgenogram prior to pneumothorax falls within two typical pictures: one of pneumonic consolidation, the other one similar to the benign group already described, with the difference that the lesions are nodular rather than reticular, striated, or linear, although in both cases there is a certain amount of lobar demarcation, the interlobar fissue moves much faster toward the clavicle in the benign group. Cough is a prominent feature here, quite often unproductive, irritating. Auscultation reveals distinct sonorous rhonchi, quite musical in character, typical of bronchial narrowing. By bronchoscopy I have not been able to see such advanced cases of bronchial stenosis, occlusions, and obstructions as described by some authors. A constant finding, particularly in the left side, has been a marked degree of swelling and reddening of the mucosa amounting to submucus infiltration. The opening of the upper lobe bronchus is definitely narrower, sometimes deformed to a buttonhole shape. It has to be assumed that yonder these infiltrative changes are bound to end in a considerable degree of bronchial obstruction. Bronchography readily reveals the site of this blockage (Figs. 6*A*, *B*, *C*).

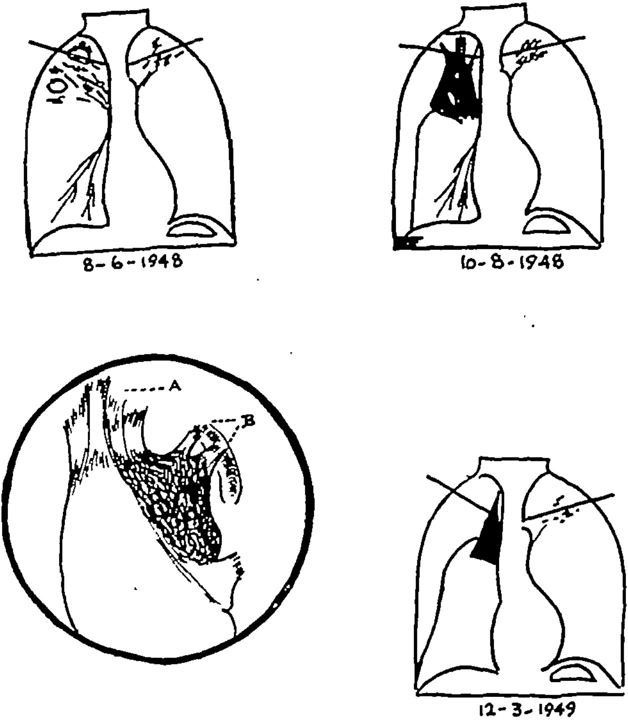
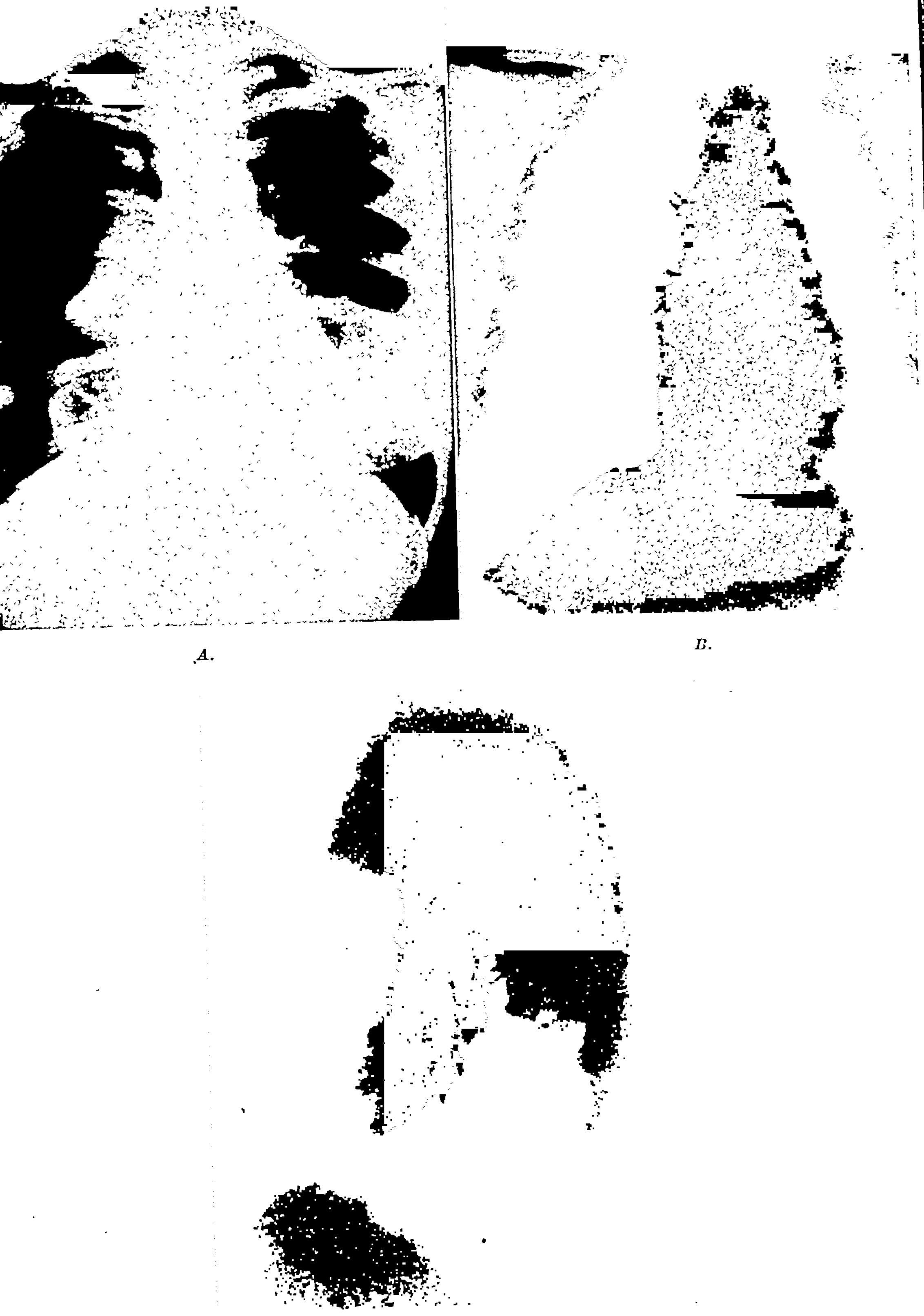


Fig. 6.—Case of beneficial "opaque lobe." Drawings reproduce appearances before pneumothorax, soon after induction, thoracoscopic view (adhesion *B* left undivided) and result following adhesion cutting.

The pathologic changes consist mainly in alveolar, interalveolar, and bronchial engorgement of fibrinous and cellular exudates, together with vascular



A.

B.

C.

Fig. 6.—*A*, case of dangerous "opaque lobe" where bronchography was done before adhesion section, this showing blockage of the main divisional branches closed to the pedicle of the lobe (Figs. 6 *B* and *C*).

dilatation. The muscular tonus and elastic properties of the lung tissue are irreparably damaged by gross toxic and necrotizing changes. Any hopes of "restitutio ad integrum" or tissue repair are here utopic unless bronchial drainage and elimination of debris assisted by good resistance and relaxation therapy (pneumoperitoneum and phrenic paralysis) can cleanse the infected lobe and thus impede the extension of the disease to other parts of the lung, to start with. A pneumothorax, and further still a "free" pneumothorax, would naturally increase the bronchial obstruction with the disastrous result

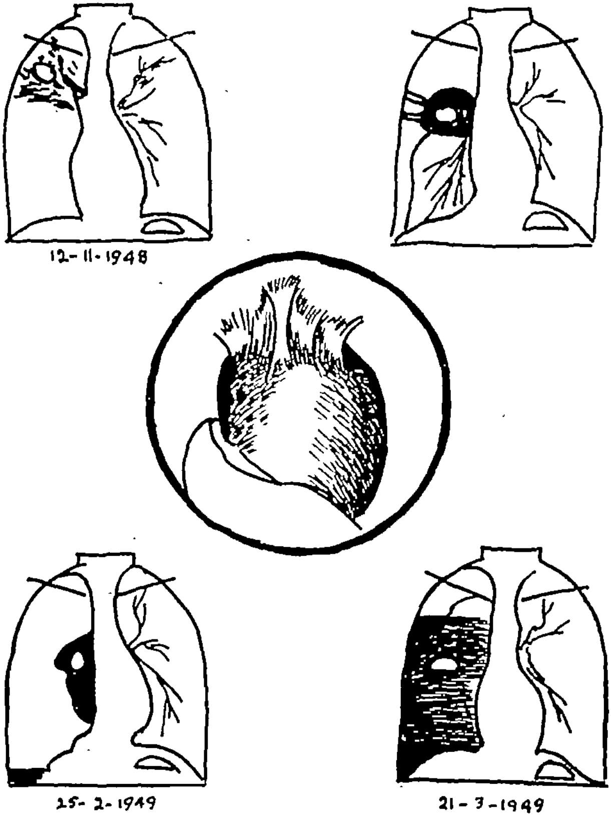


Fig. 7.—Case of dangerous "opaque lobe." Drawings reproducing appearances of roentgenograms before pneumothorax, after induction, thoracoscopic view (all adhesions were divided) and postoperative result.

of leaving highly toxic and necrotizing tissue entrapped and cut off from their natural draining channels (Figs. 7, 8, 9).

The tendency today is to submit these cases for pulmonary resection. Although by now a few hundred lobectomies have been performed for upper lobe tuberculosis, some of them for consolidated lobes, I have not been able to find

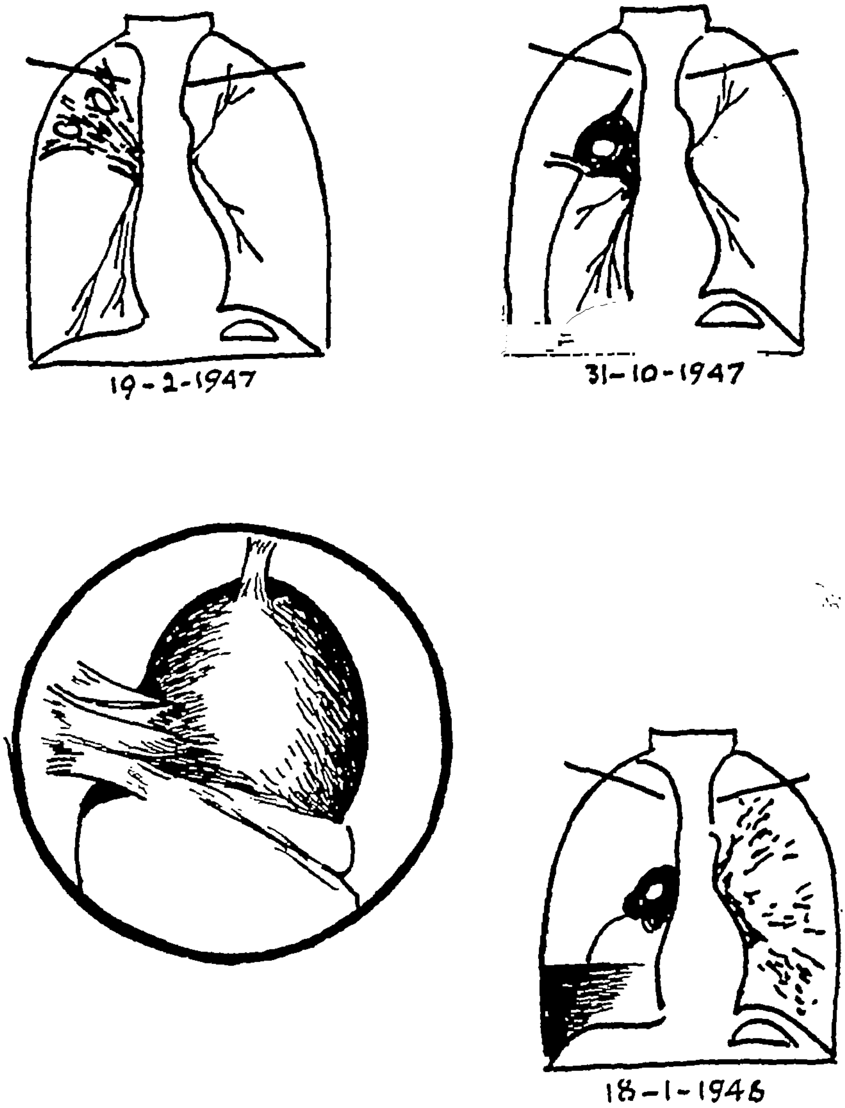


Fig. 8.—Case of dangerous "opaque lobe." Drawings reproduce radiological appearances before pneumothorax, after induction, thoracoscopic view (all adhesions were divided) and postoperative result.

enough cases to draw any worthy considerations as to the ultimate result of this form of treatment. Personally, I have followed up thirteen such cases treated by pneumoperitoneum preparatory to thoracoplasty, and the results have been, so far, highly satisfactory, definitely much superior to the results of similar cases treated by pneumothorax.

One point which is I think of considerable importance in the choice of treatment in each individual case is the general condition of the patient. A well-nourished patient, particularly of the female sex, with a triangular opaque lobe of the right upper lobe, has an almost certain guarantee of success.

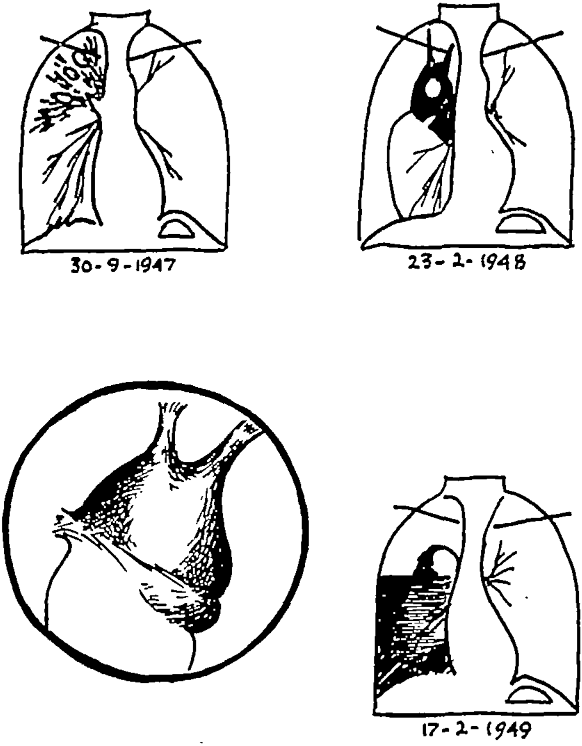

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Fig. 9.—Case of "opaque lobe," dangerous type. Drawings from roentgenograms before pneumothorax, after induction, thoracoscopic view (all adhesions were divided) and postoperative result.

SUMMARY

Opaque or "black-out" lobes in artificial pneumothorax fall within two main groups: one in which the lobe appears much decreased in size, soft, wrinkled, dark blue, light in weight, triangular (radiologically); the other where the lobe appears tense, roundish, heavy (due to consolidation) shiny, and grayish. In the first group adhesion section should consist mainly in freeing the lung from the surrounding mobile structures without aiming at complete division, To leave the opaque lobe suspended from the upper mediastinum is quite compatible with a satisfactory result, resorting, in case of concomitant diaphragmatic adhesions, to phrenic nerve paralysis if there is resulting longitudinal tension. As soon as cavity closure has been attained, the rest of the lung should be allowed to reexpand gradually by means of decreasing refills until the shrunken lobe appears cornered against the upper mediastinum giving the radiologic appearance of a collapse of the pectoral branch of the upper lobe. In these cases atelectasis can be highly beneficial, and the ultimate result comparable to selective thoracoplasty.

On the contrary, in the second group artificial pneumothorax is often very dangerous. Adhesion section seldom improves matters, and it would be safer to abandon it soon, resorting instead to a period of pneumoperitoneum and diaphragmatic paralysis preparatory to collapse or excision therapy.

These observations have been made prior to the extensive use now being made of streptomycin, P.A.S., and TRI-OM (S.32), the action of which in these types of disease seem to have had very little effect, so far, to alter substantially the results obtained prior to their use.

A table is presented with the results of 57 cases of adhesion section of opaque lobes together with considerations on their radiological, thoracoscopic, bronchographic and pathologic appearances (Table I).

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ANGIOCARDIOGRAPHIC FINDINGS IN THORACOPLASTY, ARTIFICIAL PNEUMOPERITONEUM, AND PHRENICLASIA

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IN A recent study the angiocardiographic findings in patients with artificial pneumothorax were presented.[1] These consisted of a diminution in vascularity which was directly proportional to the degree of collapse. There was crowding, distortion, and displacement of pulmonary vessels, and delay in blood flow in the collapsed lung. Varying degrees of shift of the heart, great blood vessels, and mediastinum were noted. The purpose of this report is to illustrate the angiocardiographic findings in other forms of collapse therapy commonly employed in the treatment of pulmonary tuberculosis, namely thoracoplasty, artificial pneumoperitoneum, and phreniclasia. In the past, visualization studies of the pulmonary circulation following thoracoplasty and phrenic nerve crush have been done by postmortem injection of opaque substances.[2] Whether or not autopsy findings accurately represent the changes which occur in the pulmonary circulation of the living individual has been questioned.[3] Such autopsy studies simply show the blood content of the lungs at death. Angiocardiography, on the other hand, affords an excellent method of roentgenographic visualization of the pulmonary artery and its branches in the living individual. Knowledge of the status of the pulmonary circulation in collapse therapy is important, since the circulation bears a direct relationship to the efficiency of respiration. Furthermore, changes in the circulation within the collapsed lung may play a dominant role in the healing of pulmonary tuberculosis.[4]

METHOD

For the most part, angiocardiography was conducted as originally described using a standard stereo-cassette changer permitting two films per injection.[5] Contrast substance rapidly injected into an antecubital vein passes into the right heart chambers, then to the pulmonary arteries, returning via the pulmonary veins into the left heart chambers, finally making the aorta visible. Roentgenograms were taken at appropriate intervals to visualize best the heart, great vessels, and lesser circulation. In some cases, the Fairchild automatic roll-film magazine[6] was used, enabling roentgen exposures to be made at half-second intervals during the period of opacification. Either Neo-Iopax 75 per cent[7] or Diodrast 70 per cent was used as contrast substance. Neither substance causes iodism or activation of tuberculosis, since the iodine is so bound in an organic molecule as to be unavailable to the body.[8]

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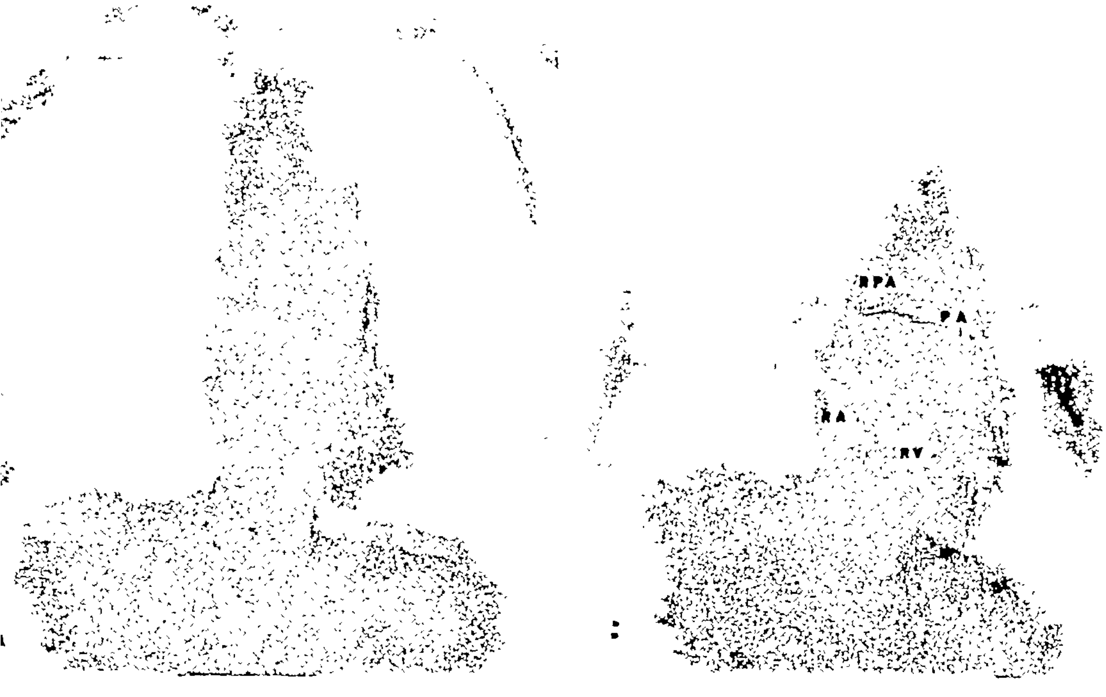
Seventeen tuberculous patients were studied at varying intervals before, during, and after collapse therapy. The types of collapse employed were thoracoplasty, artificial pneumoperitoneum, phrenic nerve crush, and a combination of the latter two. Interpretation of pulmonary vascular changes was made from comparison of the opacified pulmonary circulation of the collapsed lung with that of the noncollapsed lung, as well as from comparison of serial contrast studies in the same individual. These changes included variations in caliber, degree, and rate of filling, position, and configuration of the pulmonary arterial tree.



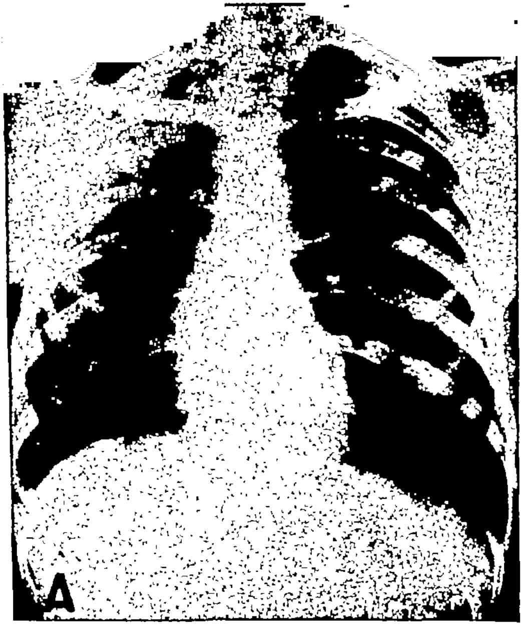
A. *B.*

Fig. 1.—(M.M. No. 182) Far advanced fibrocavernous tuberculosis of the left upper lobe in a 35-year-old woman, treated with thoracoplasty. *A.* Angiocardiogram shows the characteristic diminished vascularity of the left upper lobe. *B.* Angiocardiogram after an effective three stage thoracoplasty. Left lung volume markedly diminished. Left upper lobe vasculature diminished and pulmonary artery at left hilum appears more compact. RA=right atrium; RV = right ventricle; PA = pulmonary artery; RPA = right pulmonary artery.

FINDINGS

A definite decrease in vascularity of local areas of the lung involved by tuberculosis has been demonstrated both by angiocardiography[8, 9] and by injection studies in autopsy material.[2]

A. *Thoracoplasty.*—Following thoracoplasty there is, in addition to the avascularity at the site of the tuberculous process, a marked decrease in the vascularity of the collapsed lung roughly proportional to the degree of collapse (Figs. 1*B*, 2*C*, 3*C*). This is evidenced by crowding together, diminution in caliber, and nonfilling of pulmonary vessels. A shift of the heart and great vessels toward the noncollapsed side may occur (Figs. 1*B*, 2*C*, 3*C*). In extensive unilateral fibrosis due to tuberculosis there is retraction of the heart, mediastinum, trachea, and great vessels into the affected side. All these structures form a homogeneous shadow which on conventional roentgenograms cannot be differentiated. This may be called "fibrothorax."[10] When fibrothorax exists prior to thoracoplasty (Figs. 3*A*, 3*B*), angiocardiography may serve to delineate the great vessels and cardiac chambers usually showing rotation and displacement of these structures. Marked distortion of the heart and great vessels in this condition may be corrected to some measure by thoracoplasty. Figure 3*C* illustrates a return of these structures toward a more normal anatomic relationship following operation. Postoperatively, the pulmonary vessels on the noncollapsed side can be seen to be less widely separated, suggesting partial correction of the overdistension of this lung.

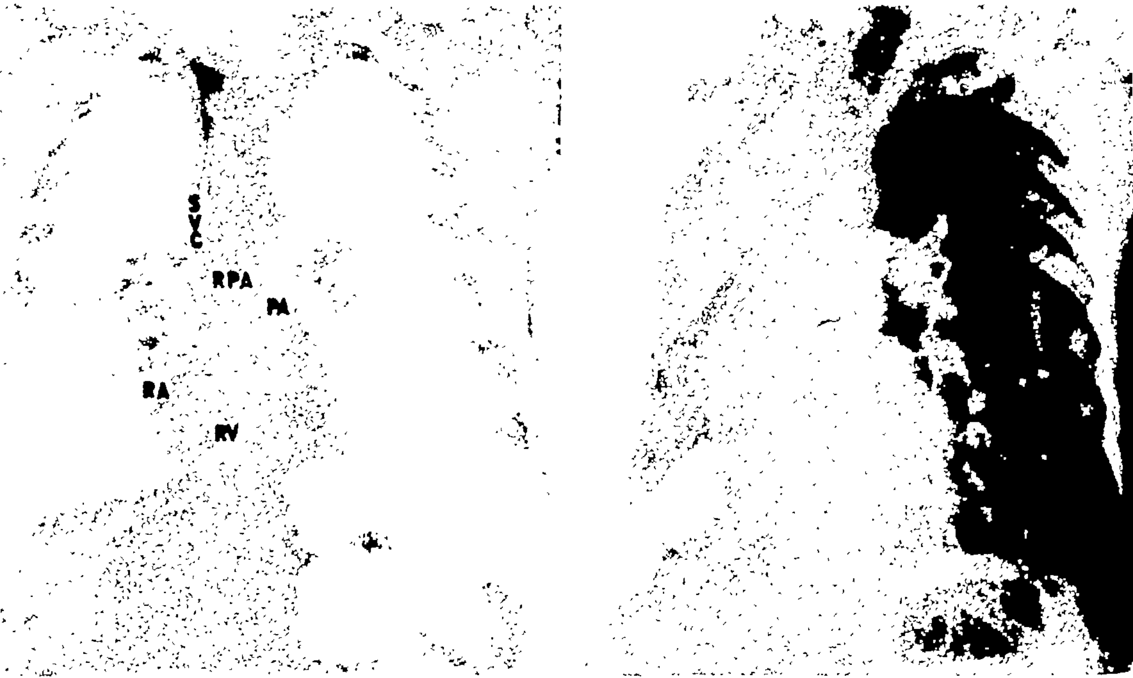


A.

Fig. 2.—(W.S. No. 243) Far-advanced fibrocaseous tuberculosis of the right upper lobe in a 15-year-old Puerto Rican treated with thoracoplasty. *A.* Conventional roentgenogram showing a 2 by 2 cm. cavity behind the first right anterior rib with bronchiogenic spread to the mid-lung field.

B. *Artificial Pneumoperitoneum.*—Visualization studies in tuberculous patients receiving artificial pneumoperitoneum revealed upward displacement of the heart and great vessels, often with cardiac rotation (Figs. 4, 5, 6). In some instances, with the patient in the erect position, the elevated right hemidiaphragm caused medial displacement of the lateral wall of the right atrium (Figs. 5, 6). The displacement appeared to be less when the elevation of the diaphragm was less as, for example, in the reclining position.

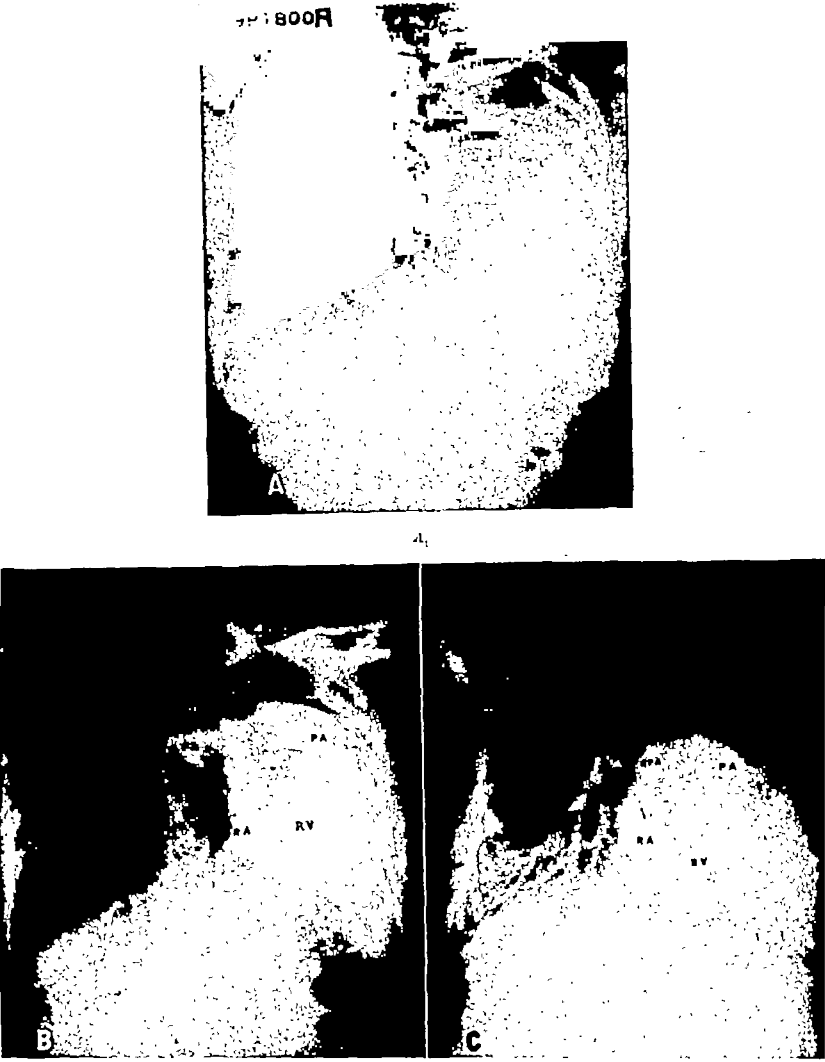
Following pneumoperitoneum the peripheral pulmonary vessels were brought closer together, and diminution and delay in opacification occurred variably. The greater the elevation of the diaphragm, the greater was the upward displacement and crowding of the pulmonary vessels. When a phrenic nerve crush was added to the pneumoperitoneum, resulting in an even greater rise of the hemidiaphragm, the vascular changes described above were accentuated on the homolateral side. Figure 6*A* and *B* not only illustrates medial displacement of the lateral wall of the right atrium, but also a narrowing of the lumen of the superior vena cava as it enters the heart. Distal to the narrowed portion there is some dilatation.



B. *C.*

Fig. 2.—*B.* Angiocardiogram five months later after preliminary collapse by an ineffective extrapleural pneumothorax. Only origin of right ascending pulmonary artery is opacified while right upper lobe is avascular. *C.* Angiocardiogram six months later after an effective three stage thoracoplasty. Right ascending pulmonary artery visualized only at orgin while right upper lobe is still avascular. The right pulmonary artery and its descending branch is compressed at the hilum.

C. *Phrenic Nerve Crush.*—Along with the elevation of the diaphragm and local avascularity associated with tuberculosis, visualization studies after phrenic nerve crush reveal some upward displacement and crowding of pulmonary vessels (Fig. 7). There may be slight elevation and rotation of the heart if the phrenic crush is left sided. A moderate diminution in vascularity and delay in blood



A_1

B.

C.

Fig. 3.—(J.D. No. 215) Fibrothorax left lung due to fibrocavernous tuberculosis in a 20-year-old white woman. *A.* Conventional roentgenogram. Extensive fibrosis of left lung with deviation of the heart, trachea, and mediastinum into the left thorax and cavities of the left upper lobe. *B.* Angiocardiography shows the position of the superior vena cava, the right heart chambers, the pulmonary artery and its branches, and reveals that the heart has assumed the right oblique position. The left pulmonary artery (seen on end) is markedly diminished in size, and there is no pulmonary arterial circulation demonstrable in the left lung. The elongation of the right pulmonary artery and the wide separation of the pulmonary arterial tree suggests overdistension of the lung. *C.* Angiocardiography after thoracoplasty shows return of the heart and great vessels toward the midline position. There is no overdistension of the right pulmonary arterial tree and the right pulmonary artery is no longer elongated.

it not seem probable that they would have attracted some attention, because they are interesting tumors, they are curious tumors? Would it perhaps be fair to make a suggestion such as Dr. Womack and I made in our article in 1937, and as Dr. Goldman has also said today, is it not a fair assumption that many of these tumors when they become wildly malignant or definitely malignant make it impossible for the pathologist to recognize that they have originated in an adenoma in the bronchus, because it is impossible to tell after they have spread all over and have involved all the organs. Now, if there were such an occurrence, what sort of tumor would we expect to find? Remember that this adenoma that Dr. Neuhof is talking about is composed of little round cells, scanty cytoplasm, almost entirely nucleus. Dr. McDonald tells us, and shows us a picture, and says that the British call the most malignant type of bronchiogenic carcinoma the oat cell carcinoma. The cell which is seen in these so-called oat cell carcinomas looks exactly like these cells which you see in the bronchial adenomas that Dr. Neuhof is describing. I will pose the question again, then. Is it possible that sometimes these tumors become so malignant that they are recognized only as so-called oat cell carcinomas but which had their origin perhaps in a so-called bronchial adenoma?

One more point which has been mentioned here this afternoon is that these adenomas are characterized by the fact that there is no ulceration in the mucosa overlying. That is true, but even without ulceration of the mucosa overlying I have seen two patients with metastases of the liver from which presumably they died. I have seen other cases with extensive lymph gland involvement. Finally, I would like to make one other statement which is not too conclusive. We have been interested in the very interesting technique which Dr. Harry Green of the Pathology Department at Yale has developed—I am sure many of you are familiar with it—of injecting pieces of tumor into the anterior chamber of the eye of guinea pigs. His contention is that no tissue will grow by such technique except two kinds, malignant tumors and embryonic tissue, and by embryonic tissue he means tissue taken from an embryo. We have recently transplanted so-called adenomatous tissue of the kind Dr. Neuhof is talking about, not the kind he dismisses as being malignant but the kind he says is benign-malignant, into guinea pigs' eyes. Unfortunately, there has not been time for me to find out just what has happened to these guinea pigs, but I had a telegram Saturday afternoon from Dr. Kemler, who has been doing this work, and he said there is a growth in the eyes but he had not yet had time to examine it. I am making that statement for what it is worth. It is not conclusive but we shall report on those findings later after we have had an opportunity to study them.

DR. OSLER ABBOTT, Atlanta.—There is no more intriguing pathology in the lung than so-called adenoma. I also feel that I am not certain what to do about it. I was brought up and trained on the theory that it should be removed by total pneumonectomy. Reports have come out that these tumors may be arrested with lobectomy alone. Our policy in Atlanta had been to do lobectomy for adenoma and if the immediately resected section shows no node involvement, the operation is discontinued at that point. We felt happy with this type of procedure until we encountered a patient a little over a year ago in whom there was an adenoma and in whom considerable lymph gland involvement was noted throughout the mediastinum. A total pneumonectomy was done; there was absolutely no involvement of the glands removed, but a careful examination of the tumor microscopically revealed several microscopic fields in which there was unquestionable transformation of this tumor from its basic cellular structure into a grade 3 squamous cell carcinoma. This experience has been repeated on three occasions since then, and in every case the adenoma has transformed itself into a squamous cell carcinoma with major areas of intact so-called "benign adenoma tissue" about it. Two other patients have been encountered in whom the tumor did not transform itself and there was very extensive invasion of the posterior chest wall on the left side. One patient had extremely extensive invasion of the major mediastinal vessels. I feel that the lesion is only "benign" in its cell structure as it stands, but it is definitely premalignant clinically and can transform into a squamous carcinoma. Whatever type an oat cell carcinoma may be, it could very well have its origin in an adenoma.

DR. CHEVALIER L. JACKSON, Philadelphia.—I remember the occasion Dr. Graham referred to; at that time I reported twenty cases encountered at Temple University Clinic. Since that time we have seen twelve more such patients. Of the original twenty, three had been treated surgically, with lobectomy in two instances and pneumonectomy in one, and seventeen bronchoscopically. At the present time those patients are status quo except for one of those treated bronchoscopically, who has just recently had a very successful lobectomy, with removal of the middle and lower lobes, not because there was progression of the tumor but because she still had recurring attacks of cough with some production due to the bronchiectasis in the lower lobe. Of the twelve new cases, seven patients were treated surgically and five bronchoscopically.

I believe that the crux of the confusion lies in the pathologic and histopathologic diagnosis. I think, as Dr. Neuhof and Dr. Rabin have said, we should eliminate the mixed tumors and cylindromas, and consider these as separate groups. It is extremely interesting to hear Dr. Rabin's view on this, since he is one of the pioneers in the study of these cases. I do not believe true adenomas have any inherent tendency to become malignant, but I do not say they *never* may become malignant. However, I think treatment depends by no means entirely on the pathology; that is, on malignancy or benignancy. Bronchoscopy is always indicated first, for study and biopsy, and for clearing up the lung. Bronchoscopic removal may be curative when the tumor is attached by a narrow pedicle; bronchoscopy will be helpful always when there is need for improved drainage. The indications for pneumonectomy or partial resection are, in my opinion, chiefly in cases where it is impossible to remove the tumor bronchoscopically, and in cases where there has been irreversible damage to the distal portion of the lung as a result of prolonged bronchial obstruction.

DR. ALEXANDER E. W. ADA, New York.—I wish to present two cases which illustrate the problem under discussion. The first is that of a 52-year-old white woman, admitted to St. Luke's Hospital, New York, in May, 1947. She had experienced recurrent hemoptyses for the past twelve years. In December, 1943, bronchoscopy was performed at another hospital and a biopsy was taken from a tumor in the left main bronchus. The microscopic sections were thought to represent adenoma, although certain areas showed definite irregularity in the arrangement of the cells. One year later two successive bronchoscopies were performed in an attempt to remove the tumor. In February, 1947, another bronchoscopic biopsy was reported as adenocarcinoma. Laminographic studies showed a tumor in the lumen of the left lower lobe bronchus, with peribronchial infiltration. Findings on routine x-ray films of the chest were essentially negative. On June 2, 1947, a total pneumonectomy was performed, and recovery was uncomplicated. On gross pathologic examination, a pedunculated rounded tumor, 2.5 by 2 cm., was found just within the opening of the left lower lobe bronchus, projecting into the main branches. The tumor had a depth of 2.5 cm. and extended outward into the surrounding lung. Two centimeters distal to the tumor there was a second one, 2.5 by 1.5 cm. in size, which was similar to the first. The lower lobe showed enlarged thick-walled bronchi and bronchioles with the appearance of bronchiectases and areas of fibrosis.

Sections through the pedunculated portion of the growth showed that toward the surface the tumor was composed of long chains and anastomosing strands of rather small epithelial cells, suggesting an origin from the basal layer of the mucosa. These had a glandular pattern, but the cells were about the same size and shape, stained very deeply and, for these reasons, did not suggest a very highly malignant growth. In certain areas, however, they had invaded the cartilage of the bronchus and here there was also considerable bone. The latter was surrounded by tumor and the cartilage was detached from the perichondrium. In these areas the lymphatics were invaded, but not the nodes. Pathologic diagnosis was adenocarcinoma of bronchus, nodes not involved.

The clinical course in this patient and the early bronchoscopic and pathologic findings tended to confirm the diagnosis of adenoma. However, over a period of years the tumor seems to have acquired some of the characteristics of malignancy. This case also illustrates the possibility that more than one tumor may be present and escape recognition.

The second patient was a 35-year-old white woman, who was admitted to St. Luke's Hospital in July, 1947. Five years before, she had been hospitalized for a virus pneumonia. Soon after, she developed occasional pain in the left chest, accompanied by cough and hemoptysis. These symptoms persisted one or two weeks at a time and recurred at about monthly intervals, usually at the time of menstruation. Four years before, bronchoscopy had been done, with the diagnosis of adenoma of the lung, although the histology was more typical of lymphoblastoma. Bronchoscopic biopsy one year later was diagnosed carcinoma, and a repeat bronchoscopic biopsy in April, 1947, was diagnosed adenoma.

On admission, the x-ray films of the chest showed no pathologic lesion or abnormality. On bronchoscopy a large papillomatous red mass was visualized, filling the opening of the left upper lobe bronchus. Microscopic diagnosis of a biopsy was adenocarcinoma. On July 15, 1947, a successful left total pneumonectomy was performed.

Pathologic examination showed the tumor to arise from the posterior wall of the upper lobe bronchus. It projected into the lumen and extended toward the hilus and downward into the lingular division. Microscopic examination of the tumor showed it fungating into the lumen, where it was composed of many solid sheets of cells with a glandular arrangement. The tumor cells infiltrated the wall deeply and extended to the cartilage which was partially destroyed. The cells were also outside the cartilage in the peribronchial tissue; however, the nodes were not involved. Pathologic diagnosis was adenocarcinoma of the bronchus; nodes not involved.

These two cases tend to confirm the experience of others, that adenoma of the bronchus may undergo malignant changes, and that certain tumors presenting similar clinical features and bronchoscopic appearance may be be malignant tumors from the onset. It is evident that a correct diagnosis cannot always be obtained from one or more bronchoscopic biopsies. Under the circumstances it would seem that the necessity of pulmonary resection should be of prime consideration and, if performed early in the disease, more conservative excisions or procedures would suffice to effect a cure.

DR. JOSEPH GORDON, Ray Brook, N. Y.—I should like to give a follow-up report on ten cases that have had a period of five years' observation. At the time of the original report by Dr. Chamberlain and myself, seven patients were reported as recovered, two died, and one was inoperable. We now have a report on six cases. Five are considered well, although one has a sinus tract infection. The sixth case is of interest to Dr. Graham. The left lobe of the liver was removed in Albany by Dr. Dickinson, and the tumor cell is identical with that of the bronchial adenoma. This patient has returned to work following lobectomy of the liver.

DR. D. H. WATERMAN, Knoxville, Tenn.—Personally, I agree with Dr. Graham and Dr. Abbott in saying that these are malignant or premalignant tumors. I would like to call attention to the bleeding that occurs on biopsy. As you know, hemoptysis is a very common symptom in the history. We have had four cases in our series in which terrific bleeding occurred when bronchoscopic biopsy was taken, one of these terminating fatally.

I believe Dr. Gordon mentioned that he had had two cases in his series where death occurred at bonchoscopy. We have been rather reluctant to treat or even biopsy these tumors through the bronchoscope when we felt resection should be resorted to anyway.

DR. W. E. ADAMS, Chicago.—A few years ago, Dr. Bloch, Dr. Steiner, and I reported on a group of these patients, and in that group there were three autopsies. One patient died from asphyxia due to obstruction of the trachea by tumor; another from pneumonia; and the third following operation. We called attention to the fact that mediastinal node metastases, and in two of the three, distant metastases to the vertebrae or liver were found. Since that time we have had experience with another patient: a 34-year-old woman who, in 1934, had a circumscribed upper lobe lesion; the sputum was negative. She was given x-ray therapy with complete disappearance of the opacity. She had no symptoms until 1942, nine years later, when hemoptyses began and the lesion appeared as in the original roentgenogram. Resection was recommended and was delayed for some months. We found no evidence of involvement of the regional lymph nodes. Eighteen months later she had a pelvic operation, and the liver was found completely studded with nodules. One of these was removed, and showed the same cellular formation as that of the original lesion. She has been completely asymptomatic since operation and shows no signs of metastases. We know, since this has been demonstrated microscopically, that she does have metastases to the liver and we do not know where else.

Experience has led us to believe that these tumors must be considered malignant; slow-growing carcinoma is a good term. From the microscopic appearance one cannot predict which of these lesions will metastasize. Thus, all should be considered potentially malignant. One cannot classify the tumor as to the rate of growth. We know that a squamous cell carcinoma may be present for years without producing symptoms.

Addendum.—This patient recently developed signs and symptoms of cerebral metastases and died shortly thereafter.

DR. EDGAR W. DAVIS, Washington.—Last night and again this morning I talked to several members of this Society about adenomas and to my amazement Dr. Graham a few moments ago used the same expression I heard last night, "We are confused." To listen to the various discussions on the management of adenomas is often more confusing. It would seem that it is virtually impossible to obtain uniform opinions from various pathologists as to the malignancy or benignancy of these lesions. It is nice to have a correct preoperative diagnosis on a patient but I do not agree that this is a clinicopathologic entity because I find from bronchoscopic biopsy and even from the surgically removed specimen that pathologists of repute differ widely in defining the nature of the lesion. It is for this reason that I doubt that the histologic report of a biopsy should necessarily dictate our mode of therapy. One pathologist may classify a given section as a grade 1 or grade 2 malignancy, whereas another pathologist may classify it as a benign lesion.

It has been our policy in the management of these so-called adenomas to take a bronchoscopic biopsy if possible. If all the tumor can be removed bronchoscopically and if all the sections by different pathologists are called benign, nothing further is done. If, however, there is any question as to whether it is a benign lesion or if it is not entirely accessible to the bronchoscope, a lobectomy or segmental resection is done. If the pathologist then reports a benign lesion it is unnecessary to sacrifice any additional lung tissue. If it is reported as questionably malignant, we do not believe anything short of a lobectomy should be done.

DR. CLARENCE CRAFOORD, Stockholm.—We have one case in which operation was done in 1936 with total pneumonectomy, which in some respects illustrates the question of malignancy of bronchial adenomas. At the time of operation the diagnosis was bronchial carcinoma. There was invasive growth in the mediastinum and metastases were found in mediastinal lymph nodes. A study later on of the specimen, however, showed that this tumor without doubt belonged to the group of mixed salivary gland tumors to which group we consider the so-called bronchial adenoma to belong. That patient was well until 1946, when he came back with recurrence in the bronchial stump and with clinical evidence of tumor growth in the mediastinum. He died about one year later. In this case there were undoubtedly clinical signs of the bronchial tumor so far back as seven years before admission. This case and many more in our experience have shown us that if tumors of this type are to be considered as potentially malignant, they cannot be clinically regarded malignant in the same sense as real carcinomas. If within a reasonable time they can be locally radically excised, the risk of a recurrence must be considered as very small. Because of that we have now come to the conclusion that it is advisable to treat these tumors more conservatively than carcinomas. As it is impossible to diagnose if there is in addition to the intrabronchial bronchoscopically visible growth also an extrabronchial extension in the great majority of cases, we are now of the opinion that endobronchial removal of the tumor is not to be con-

DR. GOLDMAN.—There is not time to answer all the problems raised. I have changed my mind from what I thought at the time I read the paper before the Society in 1938. We had never observed up to that time any lymph node metastases, and we followed some patients for twenty years. The first patient reported in that paper was operated upon about 1915, when empyema and abscess were drained. The tumor was as large as a grapefruit when the patient died. There was no lymphoid involvement.

I have modified my opinion to this extent. There are varieties of adenoma. One variety which was shown is cylindroma or mixed tumor. Of this variety shown in the first case, I have seen two cases. The *other patient also died after bronchoscopic removal of the tumor.* Early surgery with removal was not done. I believe we should differentiate the adenoma group from cancer, if for no other reason than that when we operate and remove the tumor the patient's chances are so much better, and I believe we should always separate them from carcinoma in reporting statistics.

I am somewhat pleased to find the evidence as reported by Dr. Abbott and Dr. Gordon, that these tumors may metastasize and go for many years without producing death. It seems to me they metastasize whether they are old or young. It seems local lymph node metastasis does not always occur, but is perhaps more often found in older tumors. Regarding what Dr. Crafoord said, I reported a case in 1947 which had primary bronchoscopic removal, and after three years there was local bronchial recurrence. When a lower left lobe resection was done a tumor nodule could be seen inside and outside the bronchus. This patient has had symptoms for twenty-five years and there is no sign of metastases. If in a given case all the tumor can be removed by bronchotomy, local surgical resection, there are patients in whom that can be done to conserve the lung. Regarding Dr. Eggers' cases I do not agree completely that local removal is always successful. I have seen deaths from attempt at bronchoscopic removal. I am sure every bronchoscopist is alarmed by the large amount of bleeding which may occur. Particularly where there is suppuration, opening the bronchus will not cure that patient any more than a chronic abscess, and resection should still be done for the original disease.

There is much more to be said about his subject. The ideas of the origin, and the ideas we have as to the growth of these tumors may be modified. Dr. Nathan Friedman has an idea about mixed tumors. He considers that these tumors, arising from the original cell, may form two or more different cells—and they do not have to go through a complete life cycle to complete malignancy—they may die out; thus some adenomas may develop to a certain point and go no further, and perhaps others go to complete malignancy.

DR. RABIN.—In this prolonged discussion it has been emphasized that certain members have been left with a feeling of confusion. I may say this: where confusion begins, this is the beginning of clarification. Years ago this tumor was reported simply as carcinoma and therefore did not constitute a special problem. Dr. Harry Wessler, to whom belongs the credit for recognition of the tumor, was confused when the pathologist reported several such tumors of long standing in young women as malignant. Dr. Mandelbaum admitted the growths were peculiar and different from carcinoma, and he said: "If it is shown to me that they run a benign course, we must consider them benign, even though to me as a pathologist they appear malignant."

We have passed a certain phase in the confusion regarding these growths, that is, confusion with ordinary forms of carcinoma. We have not passed the point of confusion of the adenoma with other unusual tumors. A few slides will illustrate this: (Slides.) The type shown on the screen falls into the group called the carcinoid type of adenoma which, together with the acinar type, make up the category of bronchial adenoma. I would exclude the cylindroma, which looks like certain portions of salivary gland tumors, mixed tumors containing bone and cartilage and sometimes other elements, and the entire group of mixed tumors, including osteochondroma. In addition, tumors resembling certain basal cell carcinomas of the skin showing cystic adenomatous changes are more properly relegated to carcinoma or cylindroma and should also be excluded.

Before formulating a clinical classification of the growth one must start with a pathologic classification. The pathologist must classify what he sees microscopically, and then follow the pathologic course of the growth before he can help the clinician in planning treatment.

In this respect I wish to show the following slides. This table shows five cases with tumors in the main bronchus, and no response to endoscopic therapy. They show a history of from seven to thirty-seven years, and how much longer the tumors were present we do not know. There we have failure of endoscopic removal in patients who have a long history averaging thirty-three years of symptoms. Here we have a group of fifteen patients who appear to be well after bronchoscopic treatment and who have been followd for a considerable time, without recurrence. One has been followed for fourteen years, and five have been followed for more than ten years without recurrence being found. It must be concluded that at least some of these patients are cured by simple endoscopic removal.

We are dealing with a growth different from carcinoma. We know the history is longer; we know the sex incidence is different from carcinoma. It comprises a distinct pathologic entity. Whether this tumor is amenable to a minor form of treatment or whether it requires resection of a large amount of tissue is not basic. We must separate from the adenoma other tumors that have a definite pathologic picture relating to other growths. When we do that we find the incidence of what has been called malignant change diminishes.

I do not think we have to define malignancy. We have to know what the growth does. Here is a tumor that grows for a long time (we have one half dozen patients who have had the tumor for thirty years)—a tumor which remains separated from the remainder of the bronchial wall in most cases, but in some cases does grow through the wall and even into the lung, occasionally involving the adjacent lymph nodes, *and sometimes causes distant metastases*, the tumor tissue still maintaining a benign appearance. These are not the characteristics of carcinoma in general and calling it carcinoma makes for confusion. We do not know what term is best to attach to this growth. The word adenoma is not a good one but should be retained until a better one is found.

There is little basis for considering that many carcinomas have their origin in bronchial adenomas. Dr. Head has pointed out that the duration of life in bronchial carcinoma is uniformly short. The only type of carcinoma that has a similarity to bronchial adenoma is the small cell carcinoma. These have the shortest history and this together with the absence of a polypoid appearance to these growths excludes the possibility of their origin in a bronchial adenoma. The paucity of reports of autopsied cases of adenomas must have some other explanation than that the adenomas have changed to carcinomas by the time the patients die.

THE CLINICAL USE OF A PROSTHESIS TO PREVENT OVERDISTENTION OF THE REMAINING LUNG FOLLOWING PNEUMONECTOMY

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WHEN the chest is closed without drainage following a pneumonectomy, the pleural cavity gradually becomes filled with blood and serum. In time, this fluid may be absorbed or it clots and contracts and, as a result, the mediastinum is drawn over to the affected side. This may produce considerable overdistention of the remaining lung (Figs. 1 and 2). This phenomenon may be prevented by a thoracoplasty done electively, or to obliterate the empyema cavity in case of infection (Fig. 3).

It has now been amply demonstrated that most patients do fairly well following a pneumonectomy without a thoracoplasty in spite of emphysema of the remaining lung. Many thoracic surgeons have had the impression that patients subjected to a thoracoplasty have had a better clinical result than those who were allowed to develop overdistention of the remaining lung. Cournand and his associates[1, 2] have presented data which tend to confirm this impression. They found both in children and in adults that the exercise tolerance was better in those patients in whom overdistention of the remaining lung was prevented by thoracoplasty.

Even though the thoracic surgeon agrees that overdistention of the remaining lung following pneumonectomy is undesirable, he may hesitate to subject his patient to an additional procedure as major as a thoracoplasty. If a simpler method of preventing mediastinal shift were available, it might be more widely used.

In a preliminary report,[3] we pointed out that overdistention of the remaining lung following pneumonectomy in dogs could be prevented by filling the pleural cavity with hollow plastic balls made of methyl methacrylate (Lucite).* In the dogs sacrificed after four months, there seemed to be so little foreign body reaction to the Lucite balls that a clinical trial appeared to be warranted. Our present report concerns observations made in dogs sacrificed one year after placing Lucite balls in the pleural cavity following pneumonectomy, as well as clinical experience with the use of this prosthesis in eight patients following pneumonectomy for carcinoma.

From the Surgical Clinic of the Hospital of the University of Pennsylvania; The Harrison Department of Surgical Research, Schools of Medicine, University of Pennsylvania; and The Department of Radiology of the Hospital of the University of Pennsylvania.

Read at the Twenty-eighth Annual Meeting of The American Association for Thoracic Surgery, Quebec, Canada, May 31, June 1, 2, 1948.

*The Lucite balls used in these experiments were obtained from the Nichol Product Company, Moorestown, N. J.

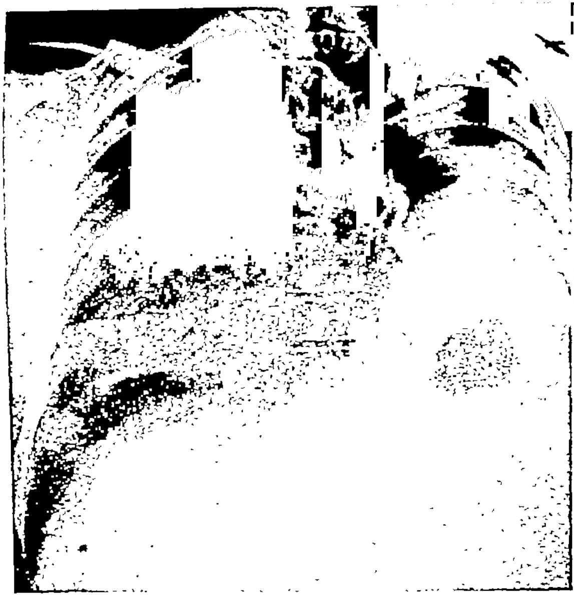


Fig. 1.

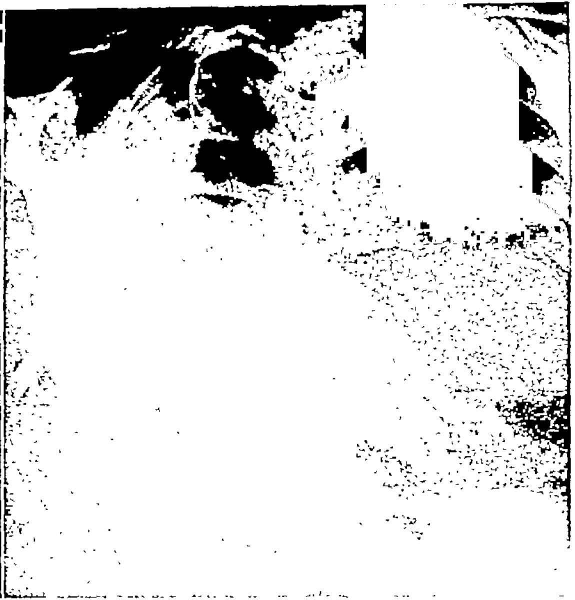


Fig. 2.

Fig. 1.—Severe overdistention of the right lung following left pneumonectomy for bronchial adenoma in a 50-year-old man. Barium in the esophagus shows it to be pulled over to the left. The right lung approaches the left side of the chest wall. (From Johnson and others: Surgery, August, 1947.[3])

Fig. 2.—Severe overdistention of the left lung following right pneumonectomy for carcinoma in a 58-year-old man. This man has had a five-year cure and complains only of some dyspnea on exertion. (From Johnson and others: Surgery, August, 1947.[3])

ANIMAL EXPERIMENTATION

As mentioned in our previous report, when no prosthesis was placed in the pleural cavity, the remaining lung became overdistended and partly filled the pleural cavity on the opposite side (Fig. 4). When Lucite balls were placed in the pleural cavity after removal of the lung, the mediastinum was maintained in the midline and overdistention of the remaining lung did not occur (Fig. 5, *A* and *B*). Since the previous report, four dogs were sacrificed one year following the insertion of the prosthesis. Four dogs remaining are living and apparently well approximately eighteen months after operation.

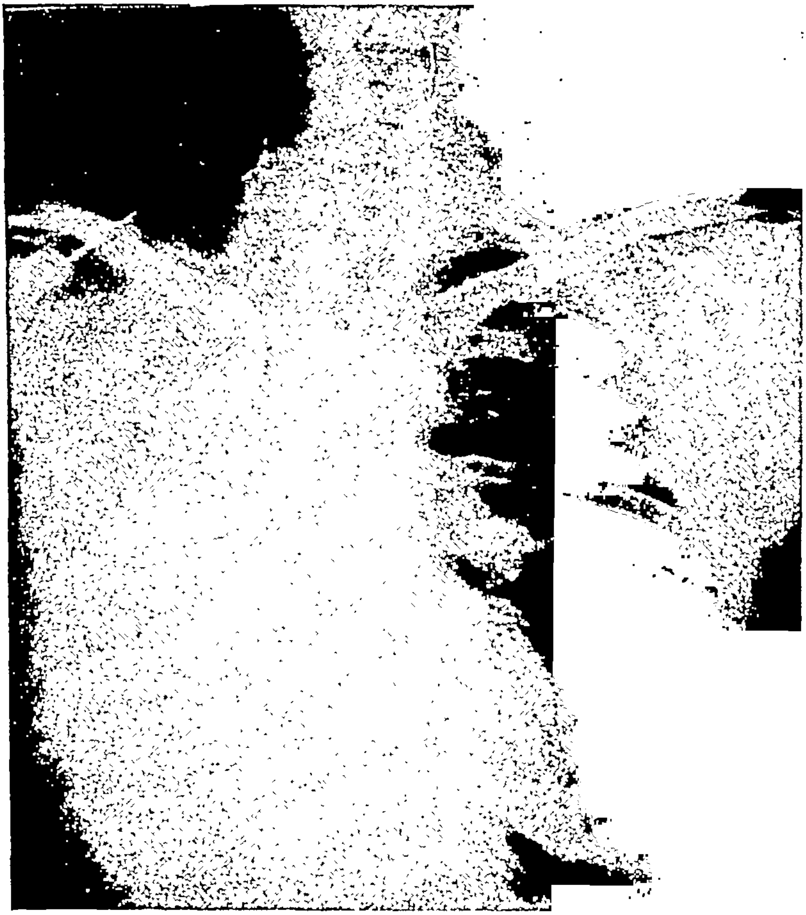


Fig. 3.—One year following right pneumonectomy and thoracoplasty for carcinoma of the lung in a 54-year-old man. The mediastinum is maintained near the midline and severe overdistention of the remaining lung is avoided. (From Johnson and others: Surgery, August, 1947.[3])

In all of the dogs the Lucite balls could be heard with a stethoscope to rattle with each heart beat. At autopsy in one dog each ball was found to be almost completely encapsulated by fibrous tissue with no fluid between the balls (Fig. 6). *As seen in Fig. 7, the balls were still in contact at some points.* Microscopic examination showed the tissue covering the balls to be acellular fibrous tissue with very little foreign body reaction (Fig. 8).

In the other three dogs, the Lucite balls were encapsulated as a group (Fig. 9). It appeared that each ball had its own niche as evidenced by the

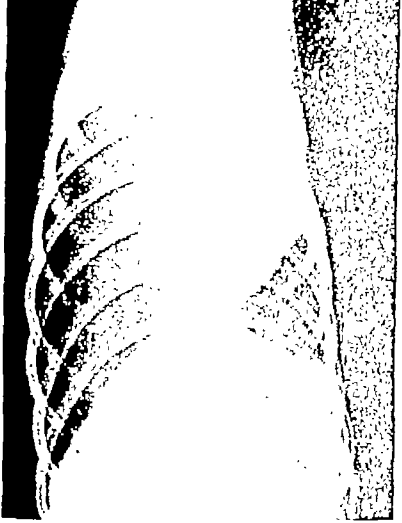
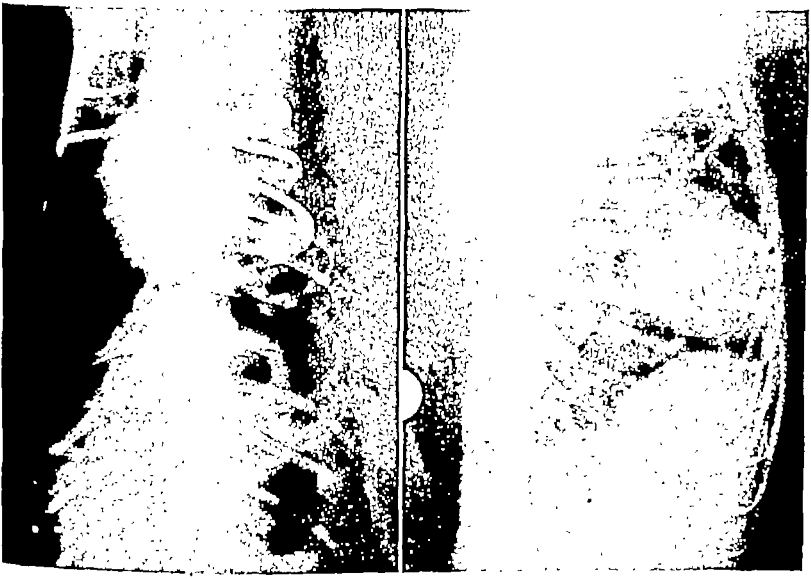


Fig. 4.—A dog two months following left pneumonectomy without the use of a prosthesis. The mediastinum is over against the left side of the chest wall and the overdistended right lung fills the lower part of the left side of the chest cavity. (From Johnson and others: Surgery, August, 1947.[3])



A. *B.*

Fig. 5, *A* and *B*.—Anteroposterior and lateral views of a dog after left pneumonectomy with twenty-four Lucite balls in the pleural cavity as a prosthesis. (From Johnson and others: Surgery, August, 1947.[3])

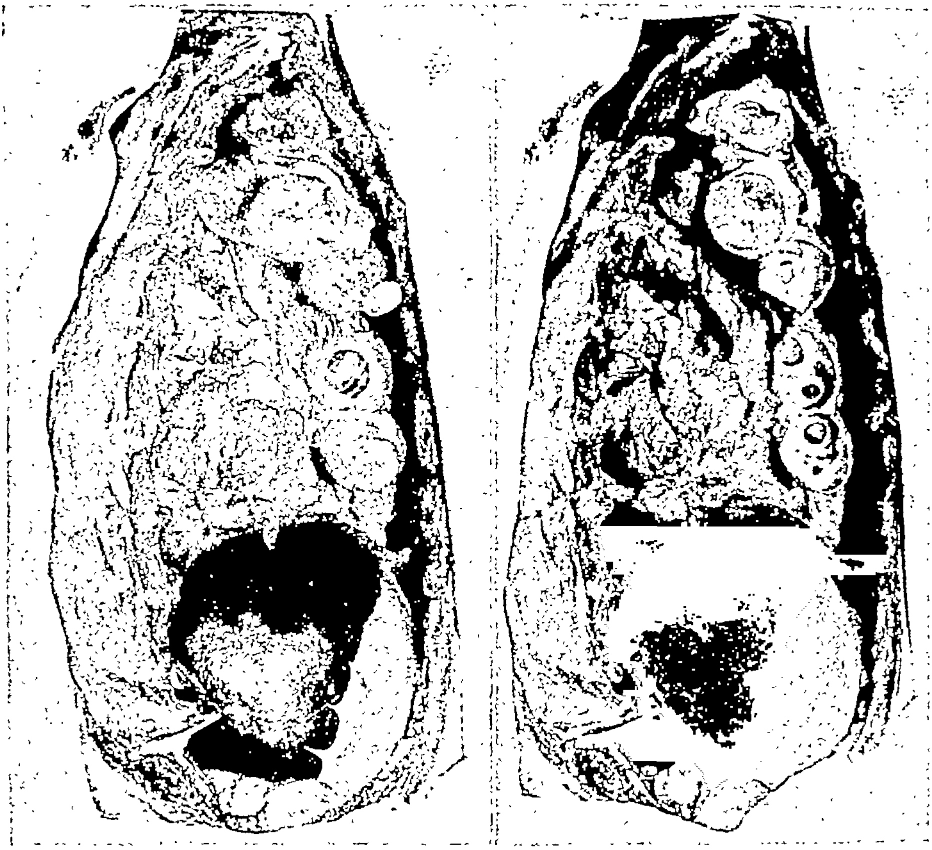


Fig. 6.

Fig. 7.

Fig. 6.—Dog sacrificed one year after pneumonectomy and insertion of Lucite ball prosthesis. In this dog the balls were almost encapsulated individually with fibrous tissue. There was no free fluid present.

Fig. 7.—Same as Fig. 6, showing that there was still contact between the balls at some points.

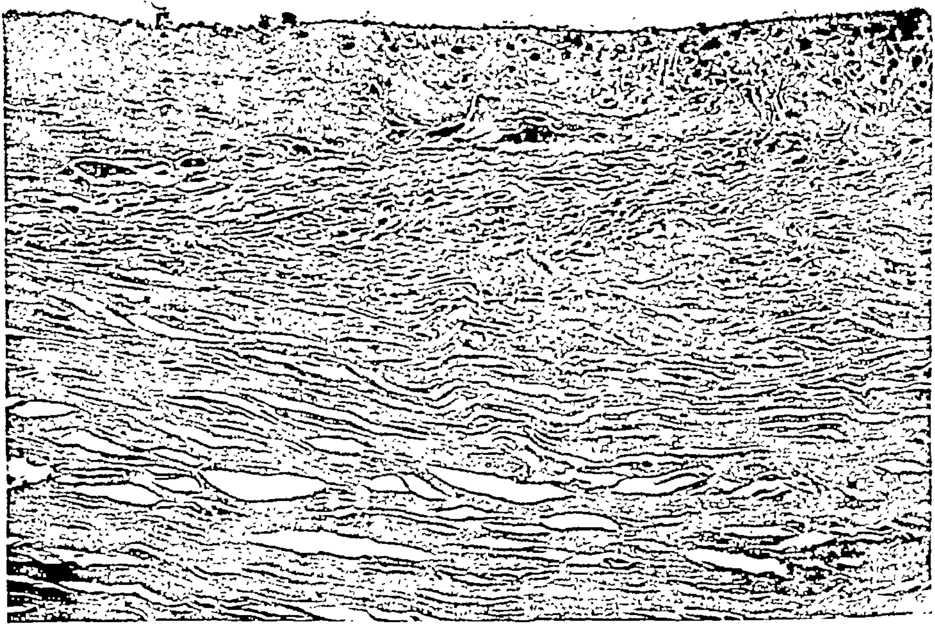


Fig. 8.—Photomicrograph of avascular fibrous tissue encapsulating Lucite balls in dog shown in Figs. 6 and 7.

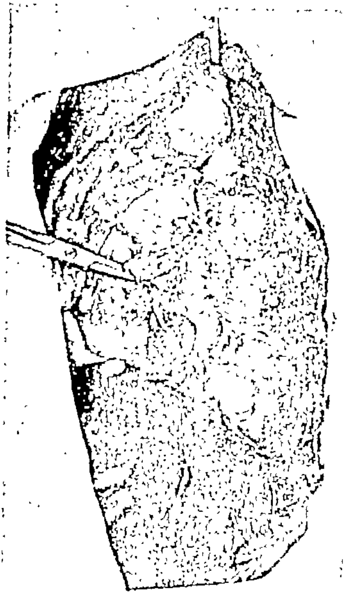


Fig. 9.—Dog sacrificed one year after pneumonectomy and insertion of Lucite ball prosthesis. In this dog the balls were encapsulated as a group. The space between the balls was occupied by clear fluid.

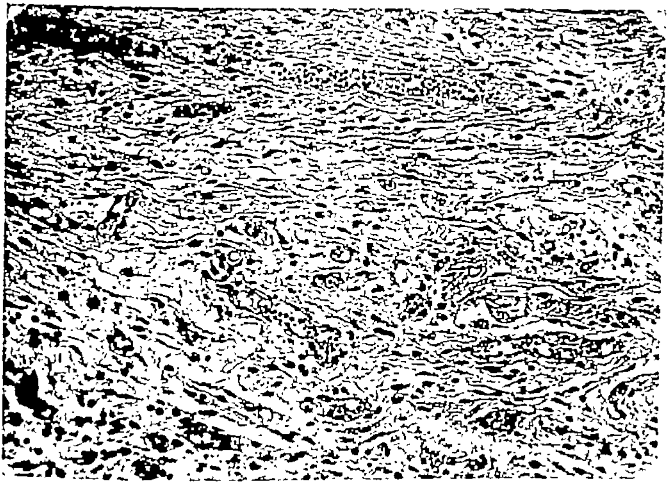
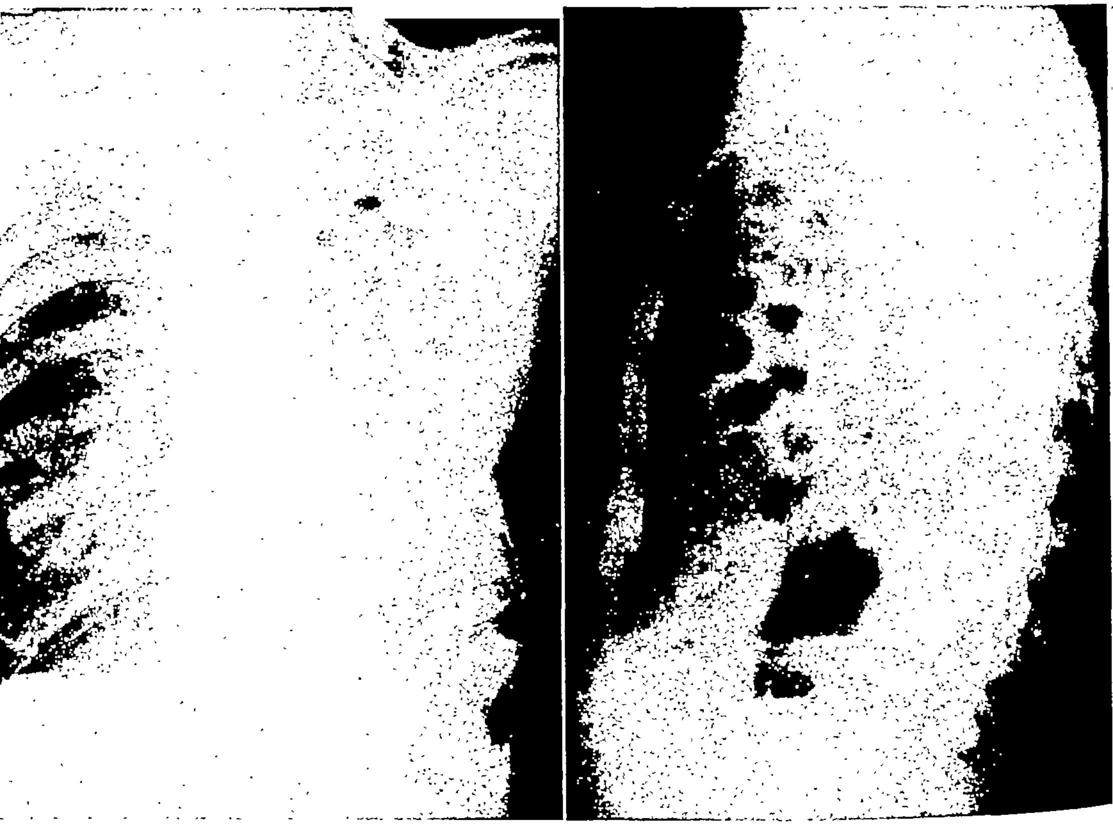


Fig. 10.—Photomicrograph of tissue removed from the pleura between the points in contact with the balls in dog shown in Fig. 9. This tissue is very vascular with cellular infiltration.

impressions on the surrounding pleura. The space between the balls was filled with clear fluid. Microscopic examination of the smooth pleura where a ball was in contact with it was similar to that in Fig. 8. On the other hand, the pleural surface between the balls had the appearance of granulation tissue (Fig. 10). The capsule surrounding the entire group of balls was thick and avascular.

A Lucite ball prosthesis seemed to be the most satisfactory of the prostheses considered for the following reasons: (1) conformity to a pleural cavity of any size or shape by varying the number of balls; (2) lack of evidence of increased infection in the pleural cavity; (3) minimal foreign body reaction; (4) ease of removal through a small incision in the event of infection; and (5) lightness of weight.



A. *B.*

Fig. 11, *A* and *B*.—Anteroposterior and lateral chest films of a patient with a Lucite ball prosthesis eight months after pneumonectomy. Seventy-five balls were used. There is no overdistention of the remaining lung.

The patients chosen for clinical trial had pneumonectomies for carcinoma. It seemed advisable to try the prosthesis cautiously at first in patients whose life expectancy was short in order to exclude possible unforeseen complications before using it in patients with a longer life expectancy.

The Lucite ball prosthesis has been placed in the pleural cavities of eight patients following pneumonectomy. Inasmuch as when a thoracoplasty is done to prevent mediastinal shift, portions of only seven or eight ribs are usually resected, the pleural cavity has not been completely filled with balls in these patients. The number of Lucite balls used has varied from thirty-five to seventy-five. The patient with seventy-five balls in the left side of the chest was operated upon about eight months ago. Recent x-ray pictures show the mediastinum to be in the midline and no overdistention of the remaining lung (Fig. 11, *A* and *B*). In a smaller individual, thirty-five balls did not fill the pleural cavity but have prevented overdistention of the remaining lung (Fig. 12, *A* and *B*).

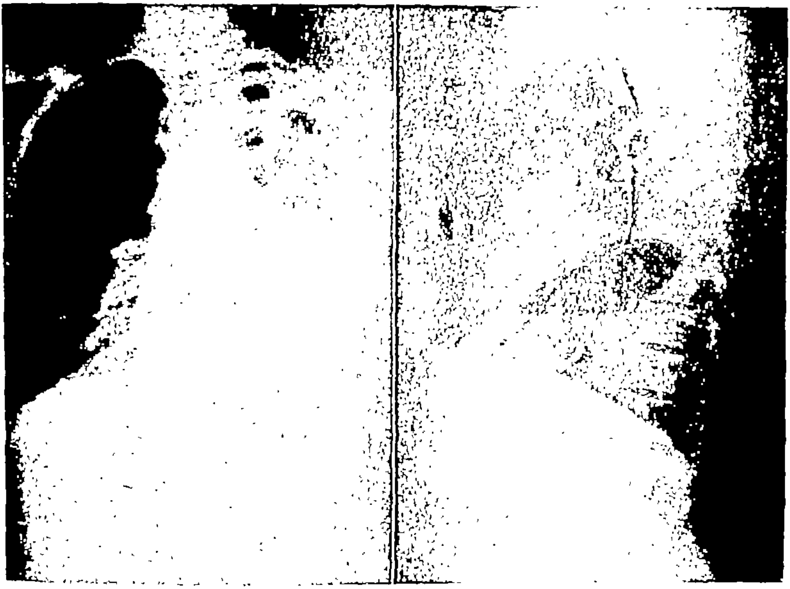

A. *B.*

Fig. 12, *A* and *B*.—Anteroposterior and lateral chest film of a patient with a Lucite ball prosthesis six months after pneumonectomy. Only thirty-five one-inch balls were used. The mediastinum is maintained in the midline.

Comment.—Immediately after operation, when the Lucite balls were surrounded by air, one could occasionally hear them rattle a foot or two away from the patient. At that stage, the patient as well often heard the balls rattle. When the air was replaced with fluid, the patients were no longer conscious of the noise. With the stethoscope, however, the rattle may still be heard with each beat of the heart in all of the patients.

There has been no evidence that the balls have caused increased postoperative pain. In two of three patients who complained of more pain than usual, we were able to demonstrate metastatic lesions. The possibility of increased pleural effusion due to the presence of the balls was considered. Thoracenteses were done whenever required to maintain the mediastinum in the midline or slightly toward the operated side. In three of the patients, thoracentesis was never required. Two of the patients were tapped only once or twice. Three of the patients required thoracentesis as late as seven to ten days after operation but it could not be determined whether the presence of the prosthesis played a part in this.

In none of the eight patients has there been any postoperative infection or unusual febrile response. In all of the patients the prosthesis has accomplished the objective of maintaining the mediastinum near the midline. Although we have been looking for possible hazards or complications resulting from the presence of the prosthesis in the pleural cavity, none has yet been apparent. Since the dogs have been observed for only eighteen months and the first patient for only eight months, late complications of foreign body reaction may yet develop.* Our experience to date suggests that the Lucite balls may prove to be an effective and safe substitute for elective postpneumonectomy thoracoplasty.

CONCLUSIONS

1. Lucite balls placed in the pleural cavity of dogs following pneumonectomy prevented mediastinal shift and were well tolerated as evidenced by autopsy studies one year after operation.
2. In eight patients in whom Lucite balls were placed in the pleural cavity after pneumonectomy the mediastinum has remained in the midline and there have been no apparent complications resulting from the presence of the balls.
3. The evidence presented to date suggests that the use of a Lucite ball prosthesis may be a simple and effective method of preventing overdistention of the remaining lung following pneumonectomy.

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2. Cournand, A., and Lester, C. W.: Pulmonary Function After Adolescence Following Thoracic Surgery, Detroit, Mich., May 29, 1946.
3. Johnson, J., Kirby, C. K., Lazatin, C. S., and Cocke, J. A.: The Use of a Prosthesis to Prevent Overdistention of the Remaining Lung Following Pneumonectomy—A Preliminary Report, Surgery 22: 179-190, 1947.

(See discussion on page 187.)

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*It has now been nineteen months since the first patient was operated upon and no complications have developed. In some of the patients the balls no longer rattle.

AN EVALUATION OF EXTRAPLEURAL PNEUMONOLYSIS WITH LUCITE PLOMBAGE

Report of Fifty-one Cases

Josiah C. Trent, M.D.,† and James D. Moody, M.D. (by invitation),
Durham, N. C., and Joseph S. Hiatt, Jr., M.D.
(by invitation), McCain, N. C.

SINCE Tuffier first performed his "pleuroparietal stripping" in 1891, the operation with modifications has been used by thoracic surgeons all over the world. To maintain the collapse effected by extrapleural separation of the parietal pleura from the chest wall, a multitude of substances has been used including air, fat, muscle, paraffin, oil, gauze, cellophane, rubber, fiber glass wool, and many others. The search for the ideal substance goes on.

In 1945 Wilson and Baker[1] found that Lucite (methyl methacrylate) in the form of spheres was inert in the extrapleural space in the experimental animal and maintained satisfactory collapse of the lung. The results of this study were sufficiently encouraging to justify clinical trial.

The first patients selected for operation were "those so-called good chronics in whom thoracoplasty was contraindicated because of low vital capacity, advanced age, bilateral cavities or those who had an unsuccessful thoracoplasty." The simplicity of the operation and the immediate good results appealed to patient and surgeon alike, and many patients refused to have any but the "ball" operation. Also, as experience with the procedure was gained, the indications for operation were broadened to include those patients in whom a thoracoplasty would have been the operation of choice.

A preliminary report by Wilson[2] of the first fifteen patients on whom follow-up study was carried out for six to ten months showed thirteen improved, one unimproved, and one dead. In spite of the early good results, however, Wilson emphasized the experimental nature of the operation and warned against excessive optimism. From January, 1946, to November, 1946, thirty-six additional patients were operated upon by members of the Duke Hospital staff. As late complications began to occur the operation was discontinued in January, 1947, until adequate follow-up studies could be made and the procedure evaluated. This report is based on an analysis of fifty-one cases of extrapleural pneumonolysis with Lucite plombage in white patients operated upon one year and six months to two years and ten months prior to this report. We feel that it is highly important to report our experiences with the operation at this time, for the practice of inserting Lucite spheres in the chest is spreading and may go far before attention is called to its dangers.

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Read at the Twenty-eighth Annual Meeting of The American Association for Thoracic Surgery, Quebec, Canada, May 31, June 1, 2, 1948.

†Dr. Trent died on Dec. 10, 1948.

In Table I are presented the number of operative cases in each age group, their classification, and the type of operation which after a careful review of the clinical history and roentgenograms we feel was indicated in each case. Nearly all of the cases fall within the far-advanced class. All patients except three had bilateral involvement. Twelve patients had had a previous thoracoplasty. Nineteen had bilateral cavities or were suspected of having bilateral cavities. Thirteen patients were considered unsuitable for any type of surgical collapse because of age, extent of the disease, and clinical condition. In only six, in retrospect, would we have considered extrapleural pneumonolysis with plombage.

TABLE I. FIFTY-ONE CASES, THIRTY-FIVE MEN, SIXTEEN WOMEN

		CLASSIFICATION			TYPE OF OPERATION INDICATED			
AGE (YR.)	NO. OF CASES	M.A.*	F.A.†	THORACOPLASTY	THORACOPLASTY	REVISION	PLOMBAGE	NONE
20-30	10	3	7	1	7		1	2
30-40	13	1	12	5	6	2	2	3
40-50	16	2	14	5	8	3	2	3
50-60	9		9	1	3	1	1	4
60-70	3		3		2			1
Total	51	6 (11.8%)	45 (88.2%)	12 (23.5%)	26 (50.9%)	6 (11.8%)	6 (11.8%)	13 (25.5%)

*M.A., Moderately advanced.
†F.A., Far advanced.

The technique of the operation has been described in detail previously by Wilson.[2] In the present series the anterior approach was used forty-four times and the posterior approach seventeen times. Three of the patients had a two-stage unilateral procedure while four had a bilateral procedure.

COMPLICATIONS

Complications of the operation may be divided into two groups, those occurring during operation or in the immediate postoperative period, and those occurring several months to years after the initial procedure.

A summary of the early complications may be found in Table II. Thirteen patients had moderate to severe dyspnea which gradually subsided in all except four. In four patients the cavity was entered at the operating table. Only one of these patients survived. In three cases a mediastinal shift was observed. In one of these the spheres were inserted under a thoracoplasty and produced

TABLE II. OPERATIVE AND EARLY POSTOPERATIVE COMPLICATIONS (TWENTY CASES, 39.3 PER CENT)

COMPLICATION	NUMBER	PER CENT
Dyspnea (moderate to severe)	13	25.5
Cavity entered	4	7.8
Mediastinal shift	3	5.9
Spread	2	
Dissection downward of spheres	2	
Compression of superior vena cava	1	
Cardiac failure	1	
Urticaria	1	
Wound separation	1	

only a moderate shift. In the other two which terminated fatally the balls dissected caudally and into the mediastinum. In two patients a postoperative spread of the disease occurred. One patient died of cardiac failure, probably as a result of reduction of the pulmonary vascular bed. In one patient, immediately after operation marked engorgement of the neck and arm veins was noted. He was returned to the operating room and after removal of one sphere the engorgement was relieved. Trial reinsertion of the sphere produced the same engorgement. This phenomenon was thought to be due to compression of the superior vena cava. One patient on the tenth postoperative day developed a generalized urticaria associated with an eosinophilia which subsided spontaneously. In one patient the wound separated before the patient's discharge from the hospital. This patient later developed a bronchocutaneous fistula.

The late complications are summarized in Table III. The tuberculous infections of the extrapleural space were diagnosed by aspiration, the presence of a sinus, or reoperation. Four patients remained dyspneic after operation. Two patients had severe incapacitating chest pain. One of these became a narcotic addict before death; the other developed a tuberculous infection around the spheres two years after operation. In one patient roentgenograms showed the spheres protruding through the mediastinum into the opposite side.

Because of the difficulty in visualizing a cavity in the lung once the Lucite spheres have been inserted, the number of patients with persistent cavities under the spheres cannot be determined accurately even with the aid of laminography. A fluid level in one or more of the spheres is a frequent roentgen finding and may be a source of confusion in attempting to identify cavities. In three patients in whom definite cavities could be seen under the spheres, an attempt was made to remove the spheres preliminary to further surgical collapse or resection. In one case the spheres were so densely embedded that removal was impossible without endangering the patient's life. The attempt was abandoned after five spheres had been removed and the cavity had been entered. In the other two cases the spheres were removed with great difficulty. In each case tuberculous granulation tissue was present between the spheres. One of these patients had a fatal pulmonary hemorrhage a few days after operation, while the other developed a severe tuberculous wound infection. In five patients the spheres were removed because of tuberculous infection in the extrapleural space which developed two months to two years after operation. In most of these cases removal was relatively easy, but in two cases all of the spheres were not recovered. One of these patients was subjected to a second operation to recover a sphere

TABLE III. LATE POSTOPERATIVE COMPLICATIONS (SIXTEEN CASES, 31.3 PER CENT)

COMPLICATIONS	NUMBER	PER CENT
Tuberculous infection around spheres	11	21.5
Draining sinus	4	7.8
Spheres extruded beneath skin	4	7.8
Dyspnea	4	7.8
Severe chest pain at operative site	2	
Migration of spheres into mediastinum	1	

TABLE IV. RESULTS IN FIFTY-ONE CASES

AGE (YR.)	IMPROVED	UNIMPROVED	WORSE	DEAD	
				OPERATIVE	LATE
20-30	2	1	2	1	3
30-40	3	1	6	1	3
40-50	3	1	8	3	1
50-60	1	0	2	3	3
60-70	1	0	2	0	0
	10 (19.6%)	3 (5.8%)	20 (39.2%)	8 (15.7%)	10 (19.6%)
Total mortality 35.3%					

embedded in the apex of the lung which could not be found at the first operation. Four of these patients had draining sinuses and one an open wound. One patient who developed a tuberculous infection of the extrapleural space two months after operation had a wound separation and a fatal hemorrhage from the wound.

RESULTS

We have considered as improved those patients who improved symptomatically and who had negative sputum; as unimproved (two patients) those who were not made worse by the operation but who continued to have symptoms and unsatisfactory sputum examinations; as worse those with positive sputum and continued progression of the disease. The complete results are summarized in Table IV. Ten cases can be classified as improved. These will be given in detail. Twenty were definitely worse and seventeen patients died as a direct result of the operation; one patient died two years after operation from coronary occlusion (no autopsy). The corrected late results in forty-two cases are summarized in Table V. Excluded from this table are eight operative deaths and one recovery in which no spheres were inserted following entry of the cavity.

Eight cases in which death occurred from seven to sixty days postoperatively are considered operative deaths. All these patients were poor operative risks, four of them hopelessly ill. In three of these cases the cavity was entered and no spheres were inserted. Death resulted from infection and respiratory failure. In three cases the spheres were removed six to eight days after operation because of marked dyspnea. One patient died as the spheres were being removed on the tenth day. Another patient died of circulatory failure attributed to a reduction in size of the pulmonary vascular bed.

Of the ten late deaths which occurred from two months to two years postoperatively, four resulted from respiratory failure, one from hemorrhage from

TABLE V. CORRECTED RESULTS IN FORTY-TWO CASES*

AGE (YR.)	IMPROVED	UNIMPROVED	WORSE	DEAD
20-30	2	0	2	3
30-40	3	1	6	3
40-50	3	1	8	1
50-60	1	0	2	3
60-70	1	0	2	0
	10 (23.8%)	2 (4.7%)	20 (47.6%)	10 (23.8%)

*Eight operative deaths and one recovery in which no spheres were inserted following entry of the cavity are excluded.

a wound which had opened as a result of tuberculous infection in the extrapleural space, one from a pulmonary hemorrhage after an operation for removal of the spheres, one from respiratory failure and progression of the disease, and one from a coronary occlusion.

Of the fifty-one cases, fourteen were considered to have definite bilateral cavitation while five others were suspected of having cavities on the opposite side. Of this group with bilateral cavities, only four had a bilateral procedure. One of these patients was considered improved (Case 4), one died, and the other two had persistent cavities under the spheres. Bilateral procedures were not done in the remainder, with the exception of one case, because of death or dyspnea. One patient diagnosed by laminography as having bilateral cavities had a unilateral procedure only (Case 1). The sputum converted and is reported to have remained negative since operation.

Eleven patients had had a previous thoracoplasty and one a bilateral thoracoplasty. Of these, seven had spheres inserted under the thoracoplasty. Two of these improved (Cases 1 and 2). Of the five who had spheres inserted on the opposite side, one improved (Case 10). Five of the twelve patients died and two became worse.

Of the six patients in whom we thought extrapleural pneumonolysis with plombage was indicated as the primary procedure, three improved, one had a tuberculous wound infection, and two died.

Bronchoscopy was done on six patients before operation and only one had findings suggestive of endobronchial tuberculosis. An attempt was made in our follow-up study, however, to do bronchoscopy on all patients who would consent. Of the eighteen patients on whom bronchoscopy was carried out, to date, twelve had ulcerative tuberculous tracheobronchial disease.

No effort has been made to draw conclusions from vital capacity studies. We have found the recorded figures to be inconsistent and frequently unreliable and have therefore excluded them from the study.

IMPROVED CASES

CASE 1.—J. B., a 49-year-old white man, had a bilateral three-rib thoracoplasty in 1942, followed by a revision on the right and removal of three more ribs in 1944. He was readmitted in July, 1945, with positive sputum. Planograms revealed bilateral cavitation and bilateral thoracoplasty. Extrapleural pneumonolysis with plombage was indicated. An anterior procedure was carried out on the right with insertion of fifteen spheres. Despite the fact that cavitation was present on the left, a bilateral procedure was not done since the sputum converted after the first operation. He has since been discharged from the sanatorium and is now working but has had no recent roentgenograms. The sputum is reported to be consistently negative on culture.

CASE 2.—C. L., a 45-year-old white man, had a two-stage, six-rib thoracoplasty in 1944. The cavity remained open. He was admitted to Duke Hospital in 1945 for revision. Classified as: right thoracoplasty with residual cavity, left F.A. A revision of the thoracoplasty was indicated. In July, 1945, anterior extrapleural pneumonolysis was performed under the thoracoplasty with insertion of sixteen spheres. Five months later, because of a persistent cavity, thirteen additional spheres were inserted through a posterior incision. Subsequent roentgenograms did not show a cavity and there was no progression of the disease. The patient had one positive sputum examination in March, 1947, but subsequent cultures were negative. The patient is now working as handy man in a Chicago hospital.

CASE 3.—W. H., a 42-year-old white man, was admitted to Duke Hospital in November, 1945, with cavitation on the right. Classified as: right F.A. with cavity, left M.A. A thoracoplasty was indicated. Through a posterior thoracoplasty incision the third and fourth ribs were removed, an apicolysis was performed, and an unknown number of spheres were inserted. The first two ribs were left intact in order to prevent deformity of the chest and to prevent pressure on the nerves and vessels by the spheres. A second stage was performed and the fifth, sixth, and seventh ribs were removed. The sputum was positive on discharge from the sanatorium in November, 1947, but subsequent specimens were negative on smear and culture. Clinically he is doing well. He refuses bronchoscopy.

CASE 4.—V. K., a 32-year-od white woman, was admitted to Duke Hospital in November, 1945, with roentgen evidence of bilateral cavitation. She had had one positive gastric washing in 1942, but had never had a positive sputum. Classified as: right F.A. with cavity, left M.A. with cavity. In this case we should have recommended thoracoplasty on the right with extrapleural pneumonolysis later on the left with paraffin or oil plombage. She had bilateral anterior procedures with the insertion of fifteen spheres on each side. Discharged from the sanatorium one year ago, she is now beginning to work. Bronchoscopy in February, 1948, revealed no endobronchial tuberculosis. Sputum has never been positive. Roentgenograms showed no progression.

CASE 5.—K. Y., a 61-year-old white woman, was admitted to Duke Hospital on Dec. 20, 1945, with cavitation in the right apex. Classified as: right F.A. with cavitation, left M.A. A thoracoplasty was indicated. An anterior procedure on the right was carried out with insertion of twenty-seven spheres. She improved clinically but is slightly dyspneic and continues to have a moderately productive cough. Single sputum specimens on two occasions were negative on culture.

CASE 6.—M. E., a 21-year-old white man, was admitted to Duke Hospital in February, 1946. Roentgenograms revealed cavitation in the right apex. Classified as: right M.A. with cavity, left M.A. A thoracoplasty was indicated. An anterior approach was done with insertion of forty-nine spheres. Subsequent roentgenograms showed no progression of the disease; there was no evident cavity. Sputum examinations have been consistently negative. The patient is now working and asymptomatic.

CASE 7.—C. P., a 21-year-old white woman, was admitted to Duke Hospital in February, 1946, for surgical collapse of a persistent cavity in the left apex. Classified as: right M.A., left F.A. with cavity. A thoracoplasty was indicated. An anterior procedure was done on the left with insertion of thirty-three spheres. She developed tuberculous meningitis eight months postoperatively, but gradually recovered with the aid of streptomycin. She is still on strict bed rest, but the sputum has been repeatedly negative on culture and symptomatically she is improved.

CASE 8.—E. M., a 42-year-old white woman, was admitted to Duke Hospital in February, 1946. Roentgenograms revealed a cavity with a fluid level in the right apex. Classified as: right F.A. with cavitation, left M.A. A thoracoplasty was indicated. An anterior procedure was performed, but the number of spheres inserted was not recorded. Sputum remained negative on smear and concentrate since discharge from the sanatorium one year ago and is now asymptomatic and doing regular housework.

CASE 9.—E. D., a 58-year-old white woman, was admitted to Duke Hospital in February, 1946, with cavitation on the right. Classified as: right F.A. with cavity; left clear. A thoracoplasty was indicated. An anterior procedure was performed with the insertion of forty-two spheres. Follow-up was done at the Florence-Darlington Sanatorium, Florence, S. C., and the patient is reported as "clinically well and sputum negative."

CASE 10.—P. M., a 31-year-old white man, had a two-stage thoracoplasty on the left in 1943, followed by an anterior stage in 1945. Roentgenograms on admission at Duke Hospital in May, 1946, revealed a cavity in the right apex. Classified as: right F.A. with cavity, left thoracoplasty. Planograms revealed no evident cavitation on the left. Extrapleural pneumonolysis with plombage was indicated. An anterior procedure was done on the right; seventy-two spheres were inserted. He had some dyspnea after operation. Planograms in December, 1947, revealed no evident cavitation. Sputum was negative on culture. The patient is now at home on limited exercise. He refuses bronchoscopy.

COMMENT

Extrapleural pneumonolysis with plombage is a desirable operation because it can be done in one stage, it is technically easy in the majority of cases, the patients tolerate it well, there is no deformity of the chest, the function of good lung tissue is maintained, and the hospital stay is short.

It is not surprising, therefore, that repeated attempts have been made to find the ideal material for insertion in the extrapleural space to maintain pulmonary collapse. It is not surprising also that these attempts have failed regardless of the type of material used, largely because of the susceptibility of the extrapleural space to infection; but of the many substances employed for this purpose, air,[3] oil, and paraffin[4] have been the most successful; their use, however, is limited to highly selected cases in which thoracoplasty is not indicated. Experimental studies at first seemed to indicate that Lucite spheres approached the ideal plombage substance because of their ease of introduction and their inertness in the body, but clinical trial has failed to substantiate the early promise of the operation.

Attention has been called in a previous report[1] to the dense hyaline tissue which forms around the spheres in the experimental animal. We found this to be true also in three clinical cases without preoperative evidence of infection in which we electively removed or attempted to remove the spheres from the extrapleural space. Since Lucite has been shown to be an inert material in the body the probable explanation for this phenomenon lies in the spherical shape of the plastic material which allows sufficient space between the spheres for serum to accumulate, clot, and organize. Once this dense hyaline mass has formed it is very difficult to remove the spheres it encases unless infection and liquefaction of the surrounding tissue occur. Removal of the spheres post mortem in one case required a mallet and chisel. It appears, therefore, that Lucite plombage in some cases must be a definitive procedure. If it is not successful in a given case, frequently no other procedure, such as thoracoplasty or resection, is feasible since the success of such an operation is dependent upon removal of the spheres.

Early results of the operation of Lucite plombage as performed at Duke Hospital[2] were encouraging but Wilson was careful to call attention to the fact that "too few cases had been completed and that too short a period of time had elapsed since operation to evaluate the results of this form of collapse therapy." The late results presented in our report emphasize the cogency of this warning when dealing with such a disease as tuberculosis.

Although our late results do not justify the continued use of Lucite spheres in the extrapleural space, it is possible that subsequent reports from other institutions may show that Lucite has, like paraffin and oil, a limited usefulness as a plombage material.

SUMMARY AND CONCLUSIONS

1. Fifty-one cases with operation a minimum of one year and six months to two years and ten months prior to this report have been presented in which extrapleural pneumonolysis with Lucite plombage was attempted or carried out. Ten patients (19.6 per cent) improved, three (5.8 per cent) did not improve, twenty (39.2 per cent) became worse, and eighteen (35.3 per cent) died.
2. Lucite plombage in some cases must be a definitive procedure, for once the spheres are inserted they are, in our experience, extremely difficult to remove unless the surrounding tissue is liquefied by infection.
3. The late results of the operation as performed at Duke Hospital do not justify continuation of the procedure.

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(See discussion on page 187.)

EXPERIMENTAL AND CLINICAL STUDY OF COLLAPSE THERAPY USING FIBERGLAS WOOL AND FABRIC

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COLLAPSE therapy is a well-established method of treatment in pulmonary tuberculosis. The simplest and most effective type of collapse therapy is artificial pneumothorax. Far from complete success is attained because of the high incidence of pleural adhesions. Even when they are completely severed by pneumonolysis, the lung may exhibit a tendency toward re-expansion. This is often initiated by the attendant pleural reaction which incites an obliterative process.

A selective type of collapse would seem to be the ideal, whereby only the diseased portion of the lung is collapsed. The undiseased portions are allowed to remain expanded in order most efficiently to carry on the functions of respiration.

True selective collapse is seen to occur in only a small percentage of cases and, at that, it is certainly not at the deliberate design of the pneumotherapist, but rather by virtue of the relative physiologic relationships existing between the several lobes. Selective collapse may be considered as occurring when the diseased lobe is in a state of peripheral atelectasis (associated with a relative bronchial hypotension), whereas, the remaining lung tissue exhibits a marked tendency to remain inflated.

When this selective condition does not prevail, collapse of the diseased areas also involves collapse of a part or all of the undiseased areas and thereby denies the patient a sizable portion of good respiratory tissue. Efforts to produce deliberate selective collapse have been made. Thoracoplasty has been used extensively with a considerable degree of safety, but this always results in a severe degree of physical deformity and the end results are not too gratifying.

Extrapleural pneumonolysis has been efficient in bringing about good anatomic selective collapse, but this procedure has been handicapped by a high incidence of space infection as well as some tendency toward re-expansion too early. Attempts to combat these complications have resulted in the introduction of a variety of space-filling agents in the belief that an obliterated space would be unfavorable for the development of infection.[1] Air, saline, oil, paraffin, body tissues, plain gauze, methyl methacrylate (Lucite), and others have been used.[2, 3, 4] Most of these agents are capable of maintaining the accomplished collapse, but each has demonstrated certain undesirable features which are invariably related to the physical, chemical, or physiologic properties of the agents used. In short, because of heaviness they produce pressure necrosis,

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soft agents tend to become liquefied, those that are unyielding result in penetration into lung parenchyma, irritating agents give rise to infection, and, as in the case of body tissues, these are too difficult to mobilize in sufficient enough quantities to be of value.

In the search for more suitable materials, two Fiberglas* products have been examined. One is a Fiberglas wool product grossly resembling raw cotton. The other is a woven fabric made from Fiberglas.

This is a material which is inorganic, nonallergenic, pliable, nonhygroscopic, noncombustible, and is light in weight, one cubic foot weighing 0.3 to 0.5 pound.†

In an attempt to evaluate the ability of animal tissues to tolerate these materials as foreign bodies over a period of time, two series of experiments were undertaken.

In one, Fiberglas wool was placed in the extrapleural space of each subject in a series of fifteen dogs. The dogs were subsequently sacrificed and specimens were taken from the implanted areas. The duration in time of these implantations ranged from one week to eight months. On gross examination, adhesions were usually noted occurring between the parietal and visceral pleura immediately adjacent to the implanted Fiberglas. Fifty per cent showed moderate to occasionally severe giant-cell foreign body reaction, and all showed either thickened pleura or fibrinous deposition, indicating considerable tissue reaction encountered in the use of raw Fiberglas wool.

The second group of experiments was carried out on fourteen dogs, in which Fiberglas fabric was applied directly to the intact pleural surfaces in the form of a bag to encompass a single lobe. First, the selected lobe was encouraged to become atelectatic, then, a sheet of Fiberglas fabric was fashioned about the lobe so as completely to contain it and prevent its re-expansion. Compression of the hilar structures was avoided. The dogs were sacrificed at intervals and specimens were obtained at ½, 1, 4, 5, and 6 months. Upon gross examination, the Fiberglas covering was found in place, undisturbed, showing no adherence to the parietal pleura. The cloth was held in close apposition to the visceral pleura by an adherence which was very fine and permitted extreme ease in removal by peeling, leaving a clean, shiny pleural surface, even at 6 months. In five unselected cases, efforts at re-expansion of these lobes were immediately successful by applying positive pressure through the dog's endotracheal tube. On microscopic sectioning of the remaining nine of these lobes, there was seen in all cases an excellent atelectasis with patent bronchi. It was usual to see a minimal mononuclear infiltration and a very mild pleural reaction. The absence of fibrosis in even the oldest specimens is indicative of a reversible condition.

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*Fiberglas; T. M. Reg. U. S. Pat. Off. OCF Corp.

†The Fiberglas superfine wool is composed of glass fibers random-oriented in a batt. Fiber diameter averages .00006 inch. The fabric, known as "116 cloth," is .004 inch thick and is of balanced Fiberglas 450-½ yarns, 60 ends per inch in warp and 58 per inch in the fill. Each strand is formed of approximately 200 individual filaments. The average filament diameter is approximately .00022 inch.

Late postoperative x-ray studies were made on five dogs and showed good collapse of the treated lobes and normal x-ray appearance of the untreated lobes.

Comparable procedures were carried out on a series of patients in order to achieve selective collapse.

CASE REPORTS

CASE 1.—N. W., a 26-year-old white woman, was admitted May 13, 1948, with a history of known pulmonary tuberculosis since June, 1943. Pneumothorax was instituted on the left at that time and the patient became symptom-free until September, 1947, at which time x-ray showed development of tuberculous involvement in the right upper lobe. Bed rest was instituted and the lesion became more extensive; a cough developed during the past six weeks. Physical examination revealed a thin, 85 pound, white woman in no distress and presenting findings of pneumothorax on the left. Chest roentgenogram demonstrated the left partial pneumothorax and also tuberculous infiltration of the right upper lobe with the presence of a central area of cavitation. On May 14, 1948, through the fifth rib bed an open pneumonolysis with Fiberglas covering was done. The patient made a rapid postoperative recovery, and smears on three occasions for acid-fast bacilli were negative.

CASE 2.—J. D., a 28-year-old white man, was admitted Oct. 13, 1947, a diagnosis of pulmonary tuberculosis having been made in 1943. The patient received right pneumothorax in 1946 and this was shortly discontinued as ineffectual. Examination revealed a fairly well-developed and well-nourished young man in no distress. There were absence of breath sounds below the fourth rib on the right, and crepitant râles were heard in the area of the scapula. X-ray showed infiltration with cavitation in right upper lobe, and free fluid in the right base. On Oct. 22, 1947, an open pneumonolysis was done through the right fifth rib bed and the upper lobe was encompassed in Fiberglas fabric. The patient made a rapid postoperative recovery and the sputum was converted to negative.

CASE 3.—H. H., a 34-year-old white man, was admitted Feb. 26, 1948, having been ill since 1943; a diagnosis of pulmonary tuberculosis had been established in 1947. Pneumoperitoneum was instituted in February, 1947, because attempts at pneumothorax were unsuccessful. The patient had 33 hemoptyses in early 1948 and was admitted in that state. Examination revealed a thin, pale, young, white man in no great distress. There were râles over both anterior apices and left posterior upper lung fields and a friction rub was heard over the latter. X-ray showed pneumoperitoneum and dense infiltration in the left upper lobe with several cavities in evidence. On Feb. 27, 1948, an open pneumonolysis was done. The upper lobe resisted satisfactory collapse because of the caseous pneumonic infiltrative process resident in the parenchyma. Collapse of the lobe was about 60 per cent, nevertheless the lobe was wrapped with Fiberglas fabric and the remaining two lobes were allowed to re-expand to fill the residual space. The patient made a rapid immediate postoperative recovery, had no recurrence of hemoptyses, and gained ten pounds in weight in the following three months. The sputum, however, continued positive. This type of patient represents a poor choice for any type of collapse therapy, and should rather have been allowed resection of the lobe.

CASE 4.—W. B., a 36-year-old white man, was admitted Jan. 15, 1947, having had a diagnosis of pulmonary tuberculosis four years prior to admission. Pneumothorax had been unsuccessfully attempted on the right. Physical examination revealed a fairly well-developed and nourished white man in no distress. X-ray revealed infiltration in the right upper lobe with cavitation, and also a dense infiltration in the left hilar region. On Jan. 17, 1947, a right extrapleural plombage was done by first lining the extrapleural space with Fiberglas fabric and then filling the resultant fabric sac with Fiberglas wool. This modification over the animal experimentation was done to protect the surrounding tissues from the irritation found to be caused by the raw or uncovered Fiberglas wool.

The patient made a rapid postoperative recovery and, although the sputum is still positive, the Gaffky count is lower and there is seen on x-ray considerable clearing of the contralateral disease. The patient is asymptomatic.

CASE 5.—J. P., a 31-year-old white man, was admitted on Oct. 24, 1947, for check-up relating to a marked increase in dyspnea and the increasing tendency to turn a blue color with exertion. Past history revealed that he had been a "sick baby" at birth, and that he had always been troubled with shortness of breath since he could remember. In recent months he had noted swelling of the ankles when doing limited light work. Physical examination showed a well-nourished and developed young man in no distress when resting. There was a marked blue cyanosis of the lips, tongue, and nail beds. There were no râles heard in the chest. The heart was enlarged and there was a systolic thrill over the entire precordium. There was a loud, grade 4, systolic murmur present. Respiratory function tests, angiocardiography, and routine x-rays strongly intimated the diagnosis of (1) pulmonary tuberculosis, bilateral upper lobe, and (2) aplasia of the right ventricle, associated with hypoplasia of the pulmonary artery, and double superior vena cava. Therefore, reasoning that a single stage operation should be sufficient to afford major relief for both the pulmonary disease and also the cardiovascular anomaly, on Dec. 19, 1947, an open pneumonolysis was done through the fifth rib on the right and a Blalock operation was done, anastomosing the right subclavian artery to the hypoplastic right pulmonary artery. The patient's immediate postoperative course was excellent. Coughing, straining, or walking no longer produced the extreme blue cyanosis—it was no longer present. However, due to bronchial hypertension, associated now with pulmonary arterial hypertension, it very soon became impossible to keep the upper lobe collapsed as was intended at the time of operation in order to control the pulmonary disease on the worse side. Therefore, on Jan. 16, 1948, an extrapleural pneumonolysis was accomplished, the space lined with Fiberglas fabric to protect the surrounding tissues, the sac filled with Fiberglas wool, and the wound closed in the usual manner with good healing. The sputum remained positive, not surprising in view of the active disease in the contralateral side. The patient was referred to a local sanatorium for a regimen of medical therapy.

CASE 6.—M. W., a 45-year-old white woman, was admitted on March 13, 1948, known to have had pulmonary tuberculosis since 1945. The patient had been on routine sanatorium care and because of the extent and nature of the lesions, intrapleural pneumothorax was judged as impossible, and she was admitted for surgical treatment. On examination there was seen a well-developed, obese (188 pounds), middle-aged woman, looking much older than her stated years, but in no distress. X-ray showed bilateral upper lobe chronic fibroid tuberculosis with multiple cavitation bilaterally, more extensive on the right. There were persistent râles heard over the left upper lung fields. On March 19, 1948, an extrapleural pneumonolysis was done through the right fourth rib and the space lined with Fiberglas fabric and then filled with Fiberglas wool, as was done in the previous cases. Table I shows the pulmonary function studies before and after this operation. The patient made an uneventful recovery, except that there was noted a too great collapse of the lung due to overfilling of the space. Thus, on April 9, 1948, the old scar was incised down to the neck of the Fiberglas

TABLE I. M. W., 45 WF

	ADMISSION	APRIL 8, 1948	MAY 8, 1948
Actual vital capacity	2,550.0 c.c.		1,250.0 c.c.
Respiratory rate (per min.)	11.0	13.0	12.0
Tidal air (liters)	0.609	0.490	0.417
Resting minute ventilation	6.7	6.3	5.01
Maximal breathing capacity	43.2	45.9	29.54
Ornsteen factor	6.4	7.2	5.8
Oxygen rebreathing bag	10.5		10.1
Carbon dioxide rebreathing bag	5.0		7.62

sac, the sac was opened, and about one-half of the previously inserted glass wool was removed. At the same time, a first stage thoracoplasty with removal of the first and second ribs was carried out on the left (contralateral) side. The patient made an uneventful recovery, so, on April 28, 1948, the thoracoplasty was completed with the removal of the third, fourth, and fifth ribs. Again, recovery was uncomplicated and Table I gives the pulmonary function figures. The patient showed no more dyspnea at discharge than she did on admission. The sputum was converted to negative.

CASE 7.—J. G., a 38-year-old man, was admitted April 9, 1948, dating his illness from 1937. A diagnosis of pulmonary tuberculosis had been established in 1938. Pneumothorax was instituted on the left. In 1941 a six-rib thoracoplasty had been done. He was admitted for surgical treatment of a reactivated lesion in the left upper lobe. Examination revealed a well-developed and nourished young man in no distress. There was evidence of a right thoracoplasty with a well-controlled underlying lung lesion. There were persistent râles heard through the left upper lung fields. X-ray showed a right thoracoplasty with no evidence of tuberculous activity. The left lung field showed evidence of a thickened pleura with tuberculous infiltration in the upper lobe. On April 17, 1948, an extrapleural pneumonolysis was done on the left. The space was lined with Fiberglas fabric and the sac filled with Fiberglas wool, in the same manner as the preceding cases. The patient made an uncomplicated and rapid recovery, and it is too early to evaluate the sputum conversion.

CASE 8.—T. C., a 59-year-old white man, was admitted Sept. 22, 1947, his third admission. A diagnosis of pulmonary tuberculosis had been made in 1938. In November, 1946, a thoracoplasty had been done on the right side. This was unsatisfactory so in February, 1947, a revision was done with the introduction of raw Fiberglas wool into the extrapleural space in the hope of gaining a better collapse. It is noteworthy that this is the only case in which the Fiberglas wool was not covered with Fiberglas fabric to protect the tissues. Soon there developed a draining and infected sinus in the revision scar. In the meantime, several attempts at pneumothorax on the left were made without success. Physical examination revealed an emaciated white man in no acute distress. There was bronchial breathing in the left middle lung fields. The right posterior thorax showed a well-healed thoracoplasty revision scar with a small draining sinus. X-ray revealed a right thoracoplasty with no apparent active disease in the right lower lobe. There were mottled densities in the left upper lobe suggestive of cavitation. On Sept. 24, 1947, the sinus tract was excised along with the previously deposited raw Fiberglas wool, which was now in the form of a fibrofatty tissue mass measuring 20 by 10 by 2.5 cm. On microscopic section there was seen extreme fibrosis with many foreign body giant cells, which contained colorless fibrils. The wound healed per primam and the patient was discharged to sanatorium care.

CASE 9.—H. I., a 34-year-old white man, was admitted April 1, 1947, pulmonary tuberculosis having been diagnosed in 1945. Pneumothorax was attempted without success on the right. Examination revealed a well-developed and nourished man in no distress. There were râles in the right upper lung fields. On April 2, 1947, an extrapleural pneumonolysis was done on the right and the space was lined with Fiberglas fabric; this sac was filled with Fiberglas wool. The patient made a rapid recovery and the sputum was converted to negative and has so remained to the present.

CASE 10.—R. S., a forty-three-year-old white woman, was admitted May 12, 1948, for surgical treatment of bilateral pulmonary tuberculosis, known as present since 1928. The patient had been treated in a number of institutions, experiencing many remissions and exacerbations. Bilateral pneumothorax, right phrenemphraxis, and pneumoperitoneum were all abandoned as unsuccessful. On physical examination, the patient was well developed and nourished and presented râles over both upper lung fields with percussion dullness in the extreme apices. Chest roentgenograms showed upper lobe involvement with suggestion of bilateral cavitation. On May 19, 1948, extrapleural pneumonolysis was done on the left, with instillation of Fiberglas fabric lining packed with Fiberglas wool in the usual manner. The postoperative course was uneventful. It was contemplated to do the opposite side within a two-week period.

TECHNIQUE

For both types of operations the patients are given a general anesthetic using the endotracheal tube and placed in the lateral horizontal position. A curved periscapular incision is made and carried through the superficial back muscles to expose the costal cage. With the scapula translated superoanteriorly, a section of the fourth or fifth rib is removed. When the use of Fiberglas cloth wrapping is intended, the pleura is opened and an intrapleural open pneumonolysis is done. The lobe is rendered atelectatic and a sufficient amount of cloth is cut to allow complete investment of the collapsed lobe. The free edge or periphery is infolded and sutured around the circumference of the lobar hilum with interrupted No. 40 cotton. This is accomplished in a gathering manner so as to provide a snug but not tightly constricting covering.

When the use of plombage is desired, an extrapleural stripping is carried out in the standard manner. Then, the space so created is completely lined with one layer of Fiberglas fabric with the neck of the cloth sac presenting at the wound. This sac is then filled without resorting to tight packing and the neck of the sac is sutured shut. In both operations a drainage tube is left in place with suction for twenty-four to forty-eight hours to prevent accumulation of fluid incident to the operative trauma. The patients have all been ambulatory within twelve to twenty-four hours, restricted only by the limits of the tubing.

DISCUSSION

The indications for these procedures would seem to be the same as for any type of lesion in which one would expect favorable response to permanent collapse therapy of a selective nature. The problem of overdistention of the uninvolved lung tissue might give concern in the use of the Fiberglas wrapping. This concern, however, should not be any greater than that appreciated in the normally occurring sequence of pathologic changes seen when lesions of any type heal. This healing is attended by fibrosis and sometimes extensive contraction which always lays open the contiguous normal lung tissue to compensatory emphysematous changes.

It is too early to evaluate the proper placement of these procedures in the treatment of tuberculosis. Other techniques have made their entry showing good promise, only to fail due to certain inherent faults, which in the course of time precipitated late complications. Caution is therefore advocated in the use of these procedures until more evidence is at hand concerning the limits of usefulness and the development of possible complications. Encouragement is gained from the early results and it is hoped that a continuing favorable follow-up will justify extension of this project.

SUMMARY

Experimental studies were undertaken using a combined series of twenty-nine dogs in which two procedures were done. In one, extrapleural Fiberglas raw wool packing was affected. This resulted in tissue reaction which was regarded as unsatisfactory. In the second series, Fiberglas fabric was used intrapleurally to invest a selected lobe. This was found satisfactory in so far as excellent selective atelectasis was accomplished, and also that the implantation of this material occasioned only a minimal tissue reaction. The case histories of ten patients were reviewed. In three, the upper lobes were wrapped in Fiberglas fabric to accomplish selective collapse. Seven had extrapleural pneumonolysis and filling of the created space with Fiberglas wool. One of these had the raw wool alone placed in the space and this resulted in the same kind of tissue reaction as seen in the dogs in which there was considerable foreign body response, and this became infected and had to be removed. The remaining six had the spaces first lined with Fiberglas fabric which was shown in the dogs to be practically nonreactive, and then the lined space was filled with the raw Fiberglas wool. No infections resulted in these and the collapse was regarded as satisfactory.

We wish to express our thanks to the Owens Corning Fiberglas Corporation, for their assistance, and to Dr. G. F. Froio for his work on the pathologic studies. We are also indebted to Dr. Martin Entine who first suggested the use of Fiberglas as a prosthetic agent.

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DISCUSSION ON "THE CLINICAL USE OF A PROSTHESIS TO PREVENT OVERDISTENTION OF THE REMAINING LUNG FOLLOWING PNEUMONECTOMY" BY DR. JULIAN JOHNSON, DR. CHARLES K. KIRBY, DR. C. S. LAZATIN, AND DR. J. A. COCKE*; "AN EVALUATION OF EXTRAPLEURAL PNEUMONOLYSIS WITH LUCITE PLOMBAGE," BY DR. JOSIAH C. TRENT, DR. JAMES D. MOODY, AND DR. JOSEPH S. HIATT, JR.†; AND "EXPERIMENTAL AND CLINICAL STUDY OF COLLAPSE THERAPY USING FIBERGLAS WOOL AND FABRIC" BY DR. THOMAS J. E. O'NEILL, DR. HECTOR P. REDONDO RAMIREZ, AND DR. ROBERT G. TROUT

DR. WILLIAM R. RIENHOFF, JR., Baltimore.—I have enjoyed the papers very much indeed, but there are two points I would like to comment on. In the first place, I think the term "compensatory emphysema" is an inappropriate one and used rather loosely. Emphysema is a pathologic condition which denotes fragility of the elastic tissue in the alveolar septa, together with rupture of alveolar walls and confluence of alveoli. This is not a compensatory mechanism but a pathologic change. What is meant, and the correct term seems to me to be used, is "compensatory dilatation" of the remaining lung following total pneumonectomy. In 1935,‡ I believe, in conjunction with Heuer and Reichert, a series of experiments were performed to prove what happened to the remaining lung following total pneumonectomy in the dog. This experimental work showed conclusively that the remaining lung undergoes a compensatory dilatation following removal of one lung which dilatation is confined to the definitive respiratory unit, composed of the ductus alveolaris, atria, and alveoli, there being no changes in the main bronchi and not the slightest evidence of the pathologic condition emphysema, as just defined.

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*See page 164 for article by Johnson, Kirby, Lazatin, and Cocke.

†See page 178 for article by Trent, Moody, and Hiatt.

‡Rienhoff, William F., Jr., Reichert, Fred L., and Heuer, George J.: Bull. Johns Hopkins Hosp. 57: 373-383, 1935.

In various autopsies, including the first case done at the Johns Hopkins Hospital by me in 1933 for fibrosarcoma of the lung (the patient drowned five years following operation), and in another instance, a woman of 70, who died six months after operation due to cerebral metastases, section of the lung at autopsy showed no evidence whatsoever of emphysema, but established unquestionably the compensatory dilatation that was found to exist experimentally. Also, there has never been any evidence of right ventricular hypertrophy following total pneumonectomy. Therefore, I think the term "compensatory emphysema" should not be used, but instead, "compensatory dilatation."

The second point that interested me particularly in Dr. Johnson's remarks was the fact that Lucite balls were used to obliterate the thoracic cavity following total pneumonectomy for malignant tumors. It is my opinion that compensatory dilatation of the remaining lung following removal of one lung for malignant tumor should not be prevented, therefore, the use of plombage is contraindicated. This may not, and probably does not, apply to total pneumonectomy for tuberculosis, because dilatation of the remaining lung might lead to a tendency to spread of the disease in that remaining lung. However, that is not true of compensatory dilatation in the remaining lung following total pneumonectomy for nonspecific infections such as diffuse bronchiectasis or infected multilocular cysts. In a series of more than 200 cases of total pneumonectomy, mainly for malignant tumor, but also for inflammatory conditions, we have not found it necessary to use any form of intrathoracic plombage and have felt that the reason our patients have had a minimal amount of impaired cardiorespiratory function is that there was a maximum opportunity for the remaining lung to undergo compensatory dilatation. The follow-up study at the present time is being continued and will be reported at a later date.

The very fact that after operation patients having a total pneumonectomy for one reason or other are somewhat short of breath even though in an oxygen tent, but later are able to breathe perfectly well and not only perform their vocations but as well their avocations, would seem to indicate that compensatory dilatation is physiologically very helpful rather than harmful. Therefore, any method employed to discourage or impair this compensatory dilatation, it seems to me, would have a deleterious effect upon the patient.

DR. H. R. DECKER, Pittsburgh.—I enjoyed these papers very much. I have had no experience with the new products used as packs after extrapleural pneumonolysis but I have had some experience with paraffin pack, a rather small series to be sure, but interesting to me because I was able to trace the cases through a period of from eight to fifteen years. In a group of 16 cases in which I used paraffin pack, for apical cavitation and bilateral tuberculosis, there are 3 patients surviving now for periods of eight, thirteen, and fifteen years, and apparently perfectly well. I will show later the slides of these cases and discuss them a little more fully. I have a feeling from hearing the paper by Dr. Trent and his co-workers that they have discarded Lucite as the medium for holding the extrapleural pneumonolysis down. I am sure the figures published through the literature with reference to paraffin pack are better. The difficulty, however, is this: Almost all figures that one reads in the literature cover a follow-up period of not more than five years. Most of them are just for immediate postoperative periods or less than three years. I am sure, therefore, that the figures as published will deteriorate as time goes on. It is so in my own small series. For instance, the series of 16 patients, in which the pack was used for tuberculosis, shows only 3 known to be living and well. Eleven are known to be dead over a period of from one to four years following the use of the pack. The cause of death mainly is progressive tuberculosis.

I would like to show two or three slides of these patients who are surviving today after the use of the pack.

(Slide 1.) The patient was not in very good physical condition and we suggested this procedure. This is the picture taken immediately after the operation.*

(Slide 2.) The man was admitted to my care following persistent hemorrhage in 1933. It seemed that the paraffin packing might be sufficient to stop the hemorrhage, and it did.

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*The second slide shows the pack after eight years, the apical cavity closed. The patient is clinically well of tuberculosis.

This patient was discharged in 1937 as being well. This picture was taken two months ago. He worked four years in Czechoslovakia on his farm and then as a prisoner in a German agricultural camp. He is now on his way to a farm in Oregon, apparently entirely recovered and having no symptoms from the pack still in place.

DR. J. B. GROW, Denver.—I was very impressed with these papers, although when we consider the operation of extrapleural pneumonolysis we must consider an operation which has not found a great place in treatment of tuberculosis. At the same time there are many instances in which there is no other procedure to be offered the patient. These were well outlined by Dr. Trent. There is no real competition between thoracoplasty and plombage in the patient whose condition is suitable for thoracoplasty. However, for the patient not suitable for thoracoplasty, plombage offers the only procedure which will satisfactorily control the disease. As Dr. Trent said, the advantages of plombage are: (1) Any portion of the complete lung may be collapsed. (2) Pulmonary function is preserved. (3) A one-stage procedure is done instead of multiple procedures. (4) The economic factors, hospitalization and expense, are reduced. (5) Following plombage there is no paradoxical motion and a decreased incidence of spread. (6) Last, and least important, plombage is not deforming.

Our experience with plombage is based on 53 cases at the National Jewish Hospital in Denver, and 26 private cases. The cases at the National Jewish Hospital are better followed, and those are the ones I will present. Of the 53 cases, 78 per cent of those over six months following operation have a negative sputum. There was one death, which occurred in a patient who had a congenital heart ailment, a high blood count, and resulted from cerebral thrombosis.

I think it is important in considering the subject, to consider the technical features, because apparently the results are largely dependent on two factors. First, the selection of cases, and second, the technique of operation.

(Slides.) The anterior approach as described by Graham and Joannides is used. An incision is made along the anterior axillary line; the second rib is resected and the cleavage plane established between endothoracic fascia and chest wall. From an anterior approach, since most tuberculous cavities are posterior, one can dissect the lung from the chest wall under direct vision, in the adherent area. The importance of thorough dissection of the mediastinum should be emphasized. As Dr. Trent pointed out, extrapleural plombage may have to be a definitive procedure unless the lung can be dissected away from the mediastinum. When the pneumonolysis includes stripping of the upper lobe from the mediastinum, it is possible to do a lobectomy or some other type of resection under the Lucite plombage, leaving the Lucite in place.

The next patient shown had a rather thorough pulmonary function study. In spite of advanced disease he had a 124 liter per minute maximum breathing capacity, which is considered quite good. Following bilateral plombage, his maximum breathing capacity was 114 liters per minute, a very slight reduction. He still has a positive sputum and is being considered for resection of the right upper lobe under the Lucite.

The next patient was a social worker for the hospital. Following a first-stage thoracoplasty there was suppurative bronchitis and spread of the disease to the lower lobe. The entire lung became atelectatic and, in spite of repeated attempts at bronchoscopic aspiration and re-aeration of the lung, the lung remained collapsed. Normal weight was 132 pounds; she was down to 82 pounds and slipping very fast in spite of streptomycin. With the experience of these previous cases it was thought possible to carry out a rather extensive procedure on a desperate risk patient. An extrapleural pneumonolysis of the entire lung was done in one stage.

To the present time, four lobectomies and one pneumonectomy have been performd subsequent to extrapleural pneumonolysis and Lucite plombage. Pulmonary resection leaving the plombage material in place is a technically satisfactory procedure. If extrapleural pneumonolysis is done, it is important to strip the lung from the mediastinum in order to make subsequent resection possible in those patients in whom the disease is not controlled by the collapse therapy.

DR. O. C. BRANTIGAN, Baltimore.—Following the work of Dr. D. Wilson and Dr. J. Johnson I have been very much interested in Lucite ball plombage in the treatment of pulmonary tuberculosis and Lucite ball prosthesis after pneumonectomy.

The report on Lucite ball plombage by Dr. Trent is extremely discouraging. It is difficult to justify his early morbidity and mortality but the late results are difficult to challenge with our present state of knowledge. I now have completed plombage on 29 patients suffering from pulmonary tuberculosis. There have been no complications nor untoward effects. The first operation was done about one year ago. I do not have statistics on end results since it is too early to consider seriously the end results.

I vary with Dr. John Grow only on technical points. The posterior approach is preferred rather than the anterior or lateral. The apex of the lung should not be freed from the mediastinum. If left adherent to the mediastinum there will be better drainage of the bronchi and the disease thus cannot be displaced into good lung tissue and remain uncollapsed.

In considering Lucite ball prosthesis after pneumonectomy, or thoracoplasty after pneumonectomy, there are two distinct problems—one is the cardiorespiratory function and the other is the flare-up of tuberculosis after pulmonary resection for tuberculosis. For patients who have had pneumonectomy for nontuberculous disease, as advocated by Dr. Rienhoff, I have done neither thoracoplasty nor prosthesis and the results have been entirely satisfactory. If cardiorespiratory difficulty arises one can do a thoracoplasty or prosthesis. In patients who have had pneumonectomy for tuberculosis the problem is different. If one waits for a flare-up of the disease, it is usually too late for treatment. An adequate thoracoplasty after pneumonectomy is a formidable procedure. In the last ten patients, through a small incision, under local anesthesia, Lucite ball prosthesis has been done four to ten weeks after pneumonectomy. The fifth such patient developed a *Staphylococcus aureus* empyema and subsequently died. The remaining nine patients have had no difficulty, but the follow-up is short since the first was done in October, 1947.

DR. STUART W. HARRINGTON, Rochester, Minn.—I am very reluctant, and am certain most of us are, to put a foreign body into the chest cavity or any part of the body, as a matter of fact, where it is to be permanent. However, there are cases in which the remaining chest cavity will require thoracoplasty, and I think it advisable that we explore these means by which we may avoid thoracoplasty to avoid overdistention or mediastinal shift which may be troublesome to the patient.

My associate, Dr. Clagett, has been particularly interested in developing a prostheses for the chest cavity following pneumonectomy. He has given me a résumé of the work he has done in this field to the present time, which, with your permission, I will read:

"Recently, Dr. Small, Dr. Grindlay, and I reported on the use of gelatin foam sponges to fill the pleural cavity after pneumonectomy. We have carried out extensive studies on animals and have used the material clinically in 20 pneumonectomies. We believe this material has considerable merit, but it is gradually absorbed and since it causes no tissue reaction or fixation, does not accomplish any permanent benefit. Experience with plastic polythene in various forms such as tubes for replacement of defects after tracheal resection, tubes for repair of stricture of the common duct, and sheets for replacement of dural defects, has demonstrated that this material causes little tissue reaction, is elastic and flexible, and can be molded or worked into various forms and shapes. We were encouraged to try the use of this material for prostheses that could be used to occupy the pleural space left after pulmonary resection. Bags, roughly lung shaped, were made of this material and were loosely filled with Pyrex glass fiber. This resulted in a soft, flexible, and very lightweight prosthesis, which has some elasticity but which will retain its shape. Such bags were placed in the empty pleural cavity after pneumonectomy in a series of dogs. Neither mediastinal shift nor pleural effusion has occurred in these animals. Pleural reaction consists of only a slight degree of thickening. Roentgenographically these bags are almost invisible.

There have been no indications of any physiologic disturbance. The postoperative course and condition were much better than in the control dogs which underwent pneumonectomy *without insertion of the prosthesis.* Exploratory thoracotomy two and three months after pneumonectomy has shown only slight thickening of the pleura and at most, only a few centimeters of clear sterile fluid.

"This study on dogs has been in progress only a few months, and while it has been going on, another series of experiments has been carried out in rats. The rats have had one lung removed and the empty space filled with a prostheses. These rats have been killed at various times postoperatively, and the pleural reactions studied. Briefly, much less fluid and exudate than usually follows pneumonectomy was found, and this fluid contains fewer cells; the pleural cavity surface and the bag are early surrounded by a soft membrane of fiber. At first the membrane is relatively thick, but within two months it is only a thin layer of fiber. The tissue is not adherent to the bag; the bag can be removed without difficulty.

"There are some technical difficulties in the manufacture of these bags in the desired shapes and sizes. We hope to be able to develop sizes and shapes to correspond to the various segments of lung as well as the entire lung, in the near future. If these prostheses work out in long-term studies on animals as well as they have thus far, we believe such prostheses may have a useful place in thoracic surgery."

As I have said, we all hesitate to use prostheses but there are occasions in which I think they may have to be used, in order to avoid thoracoplasty.

DR. A. M. VINEBERG, Montreal.—I should like to draw your attention to a procedure which we are now using at the Grace Dart Hospital, Montreal, which we think is worth while because we use a living fascial suture, or shall we say a living fascial sheet. We take a fascial sheet from the leg and sew it across the chest after depressing the apex of the lung as is done in extrapleural pneumothorax. We suture it to the fifth interspace and on the mediastinal side we suture the fascia to the lung on its mediastinal side. In this way we hope to keep the apex of the lung down, and in the few cases in which we have used this treatment, it has been successful. I should like to show you one example of this procedure.

In subsequent cases we have placed the fascial sheet directly on the apex, and in this way have produced what nature often produces, a thickened part over the lung which we wish to collapse.

DR. LYMAN BREWER, Los Angeles.—I should like to report briefly on a small series of cases in which Dr. Dolley and I have used the Lucite ball plombage with a very limited indication. We have not employed Lucite balls as a primary plombage in place of extrapleural pneumothorax, but have reserved this technique for bad risk cases where thoracoplasty had failed. In these patients although pulmonary resection might have converted the sputum, such an operation was not possible due to contralateral disease or poor general condition. This includes a group of 31 patients, over 35 years of age, who had vital capacities around 1,000 to 1,500; five had severe diabetes. No attempt was made to pick ideal cases as we were interested to see what this procedure would do. These patients have been treated in the period from the past three to fifteen months.

The technique of operation was simple. The operative thoracoplasty wound was opened, a small extrapleural space was developed, and the Lucite balls were placed in this space. We did not free the mediastinum as has been recommended earlier this morning. It is really too early to consider the results in these cases, but we have had 15 negative cultures. In analyzing our failures, we find that bronchial stenosis, tuberculous tracheobronchial disease, and cavity elsewhere in the lung have been mainly responsible for failure to convert the sputum. In addition we have gained the preliminary impression that certain giant tension cavities, and lobes almost totally destroyed by tuberculous disease, are not suitable for extrapleural Lucite pack following unsuccessful thoracoplasty. In some cases, too, it was impossible to get adequate separation of the lung. (Slides.)

The majority of the cases reported this morning, I believe, have been mainly ones in which the Lucite ball plombage has been used primarily in place of extrapleural pneumothorax. This represents a group of patients in whom the pulmonary disease was not as well stabilized as those in our group in which considerable fibrosis of the lung was present after thoracoplastic collapse. It seems to be a reasonable preliminary conclusion that the lung in which exudative disease is present with little pulmonary fibrosis does not tolerate well an extrapleural Lucite ball plombage, while the more stable fibrotic lung following thoracoplasty will tolerate such a plombage.

We believe, therefore, in cases where pulmonary resection is not feasible following unsuccessful thoracoplasty, further trial of extrapleural Lucite plombage is definitely indicated.

DR. C. W. LESTER, New York.—It has been my privilege to have been associated with Dr. André Cournand and his staff in the study of pulmonary function after pneumonectomy, particularly in children. It is our feeling that overdistention is a condition to be reckoned with and should be avoided. Our first studies one or two years after operation made us think that perhaps overdistention was not a factor where children were concerned. Several years later we restudied the same patients after they had passed the age of puberty and had become adults, and we found overdistention was something to consider and that pulmonary function was definitely less good in those who had distention than in those who did not. We therefore thought that some method should be attempted to prevent distention of the lung while these children were growing. Thoracoplasty, in a child, of course, is a very deforming operation. We had not thought of Lucite, but of course most of the operations that I have done were in infected cases. Here suppurative lungs are being dealt with, and infection in the presence of a foreign body is something to be avoided. We therefore have tried a modified form of thoracoplasty which really is not a thoracoplasty at all. In opening the chest the fifth rib is removed completely, and then the fourth and sixth are removed in their posterior halves. What we hope to obtain by this is that, as the child grows, the upper part of the chest will contract, and the rising diaphragm will prevent overdistention at least until the child reaches adult life when a thoracoplasty can be accomplished without so much deformity. We are, of course, studying these children as we go along.

(Slides.) These two cases are very recent. We cannot draw conclusions from them as yet. One was an 8-year-old who had extensive bronchiectasis of the left lung with contracture of the lung and shift of the right chest into the left side. After operation in which the fifth rib can be seen resected completely and the fourth and fifth partly resected, the heart has moved back into the right side of the chest and there is much less distention. This child was too young to attempt functional study; oxygen saturation was normal in the blood.

The other child had essentially the same condition. After operation some shift can be seen, not very much. We found on functional study the residual air was 25 per cent, which is essentially normal, and the results have been good so far.

DR. J. D. MURPHY, Oteen.—When a stone is dropped into the placid surface of a millstream, there is set in motion a series of centrifugal waves which ultimately cover the whole pond. So, when the first Lucite ball was dropped into the extrapleural space at Duke University, Oteen being geographically close to Duke was early in contact with this wave of patient enthusiasm and we succumbed. Having had some unfavorable experiences during the extrapleural pneumothorax era of 1938 to 1939, we determined to screen our cases carefully and attempted to select, therefore, only those cases we decided were good thoracoplasty risks.

(Slides.) I will give you briefly our experience. There were 22 cases in all; 14 primary operations and 8 operations following thoracoplasty in which the cavity had not been closed. There was sputum conversion in 27.7 per cent, which does not approach that of thoracoplasty. There were 3 empyemas and 3 fistulas in the primary group; 1 empyema in the secondary cases and in 2 patients migration of the balls. In 1 patient 5 balls migrated into the mediastinum. They are still there and we are unable to find them. In one patient a ball migrated up to the neck and produced pressure on the brachial plexus and subclavian arteries. That was removed. We have removed a total of 9 packs. In cases in which empyema follows and the material is liquefied, it is not difficult. In cases in which there has not been empyema each ball is enclosed in a fibrous capsule, presenting a honeycomb-like structure. These are more difficult to work with, but can be removed without too much trouble. A uterine tenaculum forceps has been used to remove several deeply embedded balls. We have had no mortality. We started these operations in September, 1946, and became so discouraged by 1947 that in June we discontinued them. We will wait awhile before other plombage materials are tried.

DR. FRANK S. DOLLEY, Los Angeles.—It has become evident in listening to the several uncorrelated papers that have just been delivered on the subject of the introduction of Lucite balls between the lung and the chest wall for the local collapse of lung tissue affected by tuberculosis, that some attempt should be made to evaluate the things that have been said of this particular form of treatment.

My associate, Dr. Brewer, has just reviewed our results in the use of the Lucite pack. From these results we are more than ever encouraged in the use of Lucite balls. In other papers the operation has been uniformly condemned. The reason for this divergence in opinion is due, I am certain, to the selection of cases.

In all our cases except one, we have established a Lucite pack following thoracoplasty. We resect a small portion of the rib immediately beneath the thoracoplastic area, and find the line of cleavage at that point. After a small pocket is obtained by digital dissection, the space is enlarged, under direct vision by use of a Cameron light. By sharp and blunt dissection the lung and parietal pleura are freed from the immediately overlying tissues. Parenthetically, it should be said that we wait from two to three or four months following a manifestly unsuccessful thoracoplasty before we introduce the Lucite balls. For, by so doing, we are certain that the parietal pleura is sufficiently solid to minimize the danger of a pulmonary slough from ball pressure. We carefully avoid freeing the apex of the lung, for we want the pressure to be entirely lateral.

For the first three or four days following this procedure, but seldom longer than one week, the patient is often conscious of the clicking of balls in the chest when he moves around or coughs. After that time, I do not believe there has been a case where a patient has been conscious of ball movement.

On two occasions we have removed the balls several weeks after insertion. One because of pressure against a bronchus, which caused an almost constant harassing cough. This was relieved after some of the balls were removed. The other occasion was because of postoperative, nontuberculous infection. In both these cases, the sputum continued negative by culture.

We have had no deaths, and no complications that we have not been able to relieve. Our laboratory reports show 50 per cent negative sputum by culture in our series. We have used the Lucite pack only in cases where thoracoplasty has proved unsuccessful, and there seemed nothing else which offered a reasonably good chance for conversion of the sputum.

So I say, as I did in the beginning, that instead of being discouraged with the results we have obtained, we look upon the Lucite pack as a definite addition to our series of surgical procedures directed toward the cure of pulmonary tuberculosis. As in the choice of any operation for this disease, the important thing is the choice of the case selected for the particular operation contemplated.

DR. ROBERT KLOPSTOCK, Jamaica, N. Y.—I should like to make two technical remarks and one recommendation. If one has to resort to any extrapleural compression therapy using air or foreign body of a proved substance, this type of procedure should be carried out only if the pleural space has been tested for firm obliteration, prior to the operation. Extrapleural plombage is recommended as an operation of minimum trauma in cases with limited reserve. The perforation likely to occur into the pleural space if it has been free prior to operation may be a very grave complication, particularly in patients with limited reserve. The other technical point that I should like to make is that the extrapleural space should be absolutely dry, tested for being free of oozing before the foreign material is placed into it. Exudate in the plombage bed can exert prohibitive pressure upon the mediastinum and by mobilizing the plombage itself creates "the floating" ineffective plombage.

The recommendation, which may sound dissenting, would be to regard the plombage as a temporary procedure. One should remove the plombage, irrespective of substance used (even if the result proved to be satisfactory) between six or eight weeks to six months after operation and to follow it up at the same time with a limited apical thoracoplasty. The plombage, a limited surgical procedure if performed with delicate care, served its purpose by creating a very selective collapse. In order to secure the permanency of this selctive collapse it seems to be safer to replace the plombage with just a limited apical thoracoplasty than to rely on the plombage itself, for the foreign material stimulates the formation of a continuously shrinking fibrous capsule around itself. The slow shrinkage can exert a considerable pressure within the capsule. The least resistant point outside of the capsule is the surface of the damaged lung toward which the late perforations are likely to occur due to the protruding of the plombage substance if the plombage has not been removed in time. Late perforations may occur from six months up to two or more years following operation.

DR. TRENT.—I should like to say in closing that certainly we agree with Dr. Decker that the longer the cases are followed the poorer are the results. Our follow-ups, I should like to remind you, although not very long, have continued for a minimum of one and one-half years, the longest approximately three years. I have one slide I should like to show you, which illustrates one type of case included in the Duke Hospital series. Some of these cases like the one Dr. Brantigan illustrated, were selective, in that the area immediately over the cavity, which is approximately under the second rib in this case, were selected for collapse and the lung allowed to remain attached to the mediastinum. (Slides.)

This was done approximately two years before this report. The pictures show the patient approximately one year after operation and immediately after operation. When the patient was discharged the sputum was negative. While at home he resumed activity, had no symptoms, and was feeling well. We considered this a good result. A few months ago he began to develop symptoms and felt he should return for check-up. He returned two months ago and the film on the right was made. The smear was found loaded with acid-fast organisms two years after operation.

AN ANALYSIS OF VARIATIONS OF THE SEGMENTAL BRONCHI OF THE RIGHT LOWER LOBE OF FIFTY INJECTED LUNGS

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INTRODUCTION

NOTWITHSTANDING the general acceptance in this country of the Jackson-Huber terminology of the bronchopulmonary segments, and in the British Empire of the somewhat differently named but corresponding zones, there seems not to have been made, as yet, a statistical study of the variations of the lower lobe segments. Brock (1946) discussed these from the standpoint of the varying amount of territory a given segment can cover and published excellent figures of some of these injected specimens. However, there still remains the task of analyzing variations in the light of an empirically established prevailing pattern, that is, of determining the typical surface area of a segment and then noting how often, and by what other bronchi, this is supplied in whole or in part. Such an analysis would seem to be a necessary preliminary to accurate localization of lesions of the lung and to surgical resection of its segments.

The present study is restricted to the segments of the right lower lobe, but a companion article (by Berg, Boyden, and Smith), dealing with the segments of the left lower lobe, together with a summary of the differences between the two lobes, is being prepared for publication.†

MATERIAL AND METHODS

Fifty fresh specimens of right lower lobes have been used in this study. The technique of injection was that employed by Scannell (1947). After the hilar region had been freed of vessels and lymph nodes, the primary branches of the right lower lobe stem were dissected from the surrounding parenchyma for a distance of about 1.5 cm. In many instances the level of primary branching of each segmental bronchus was reached also. Following partial inflation with air to clear the passages, each segment was injected with hot colored gelatin at approximately 85° C. The colored gelatin was prepared by dissolving 75 Gm. of U.S.P. gelatin and 10 Gm. of potassium iodide in 500 c.c. of tap water at 100° C and adding 4 c.c. of appropriate vegetable dye to each 100 c.c. of gelatin. Simple aniline dyes available at the grocery store were used.

Immediately after injection of each segment, the lobe was plunged into cold tap water to aid in the gelling of the segment. After two minutes it was removed from the cold bath and another segment injected. Midway through the process the intersegmental margins, as revealed by the colored masses, were drawn on the surface with India ink. This step was repeated, following the completion of the injection process, but this time the surface of the lung was carefully dried with toweling, and under an electric fan, both before and after

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†For comparable analyses of the left and right upper lobes, see Boyden and Hartmann (1946), Scannell (1947); Boyden and Scannell (1948), and Scannell and Boyden (1948).

application of the ink. In this manner the intersegmental boundaries were preserved permanently.

After drawings were made of the anterior, posterior, and diaphragmatic views of each lung, the segmental bronchi and rami were dissected in order to check and correlate the pattern of the bronchial tree with that of the surface distribution of the injected gelatin. The fact that the lungs were solid masses of gelatin made this step much more difficult than would have been the case if one were dissecting lungs preserved by the usual embalming methods, but the essential points could be determined in each case. After the tree was in full view and drawn for the record, the stem was opened and the location of the bronchial orifices noted on the drawing.

The terminology employed in designating the bronchopulmonary segments is that proposed by Jackson and Huber (1943). The numerical designation of branches is that devised by Boyden (1945).

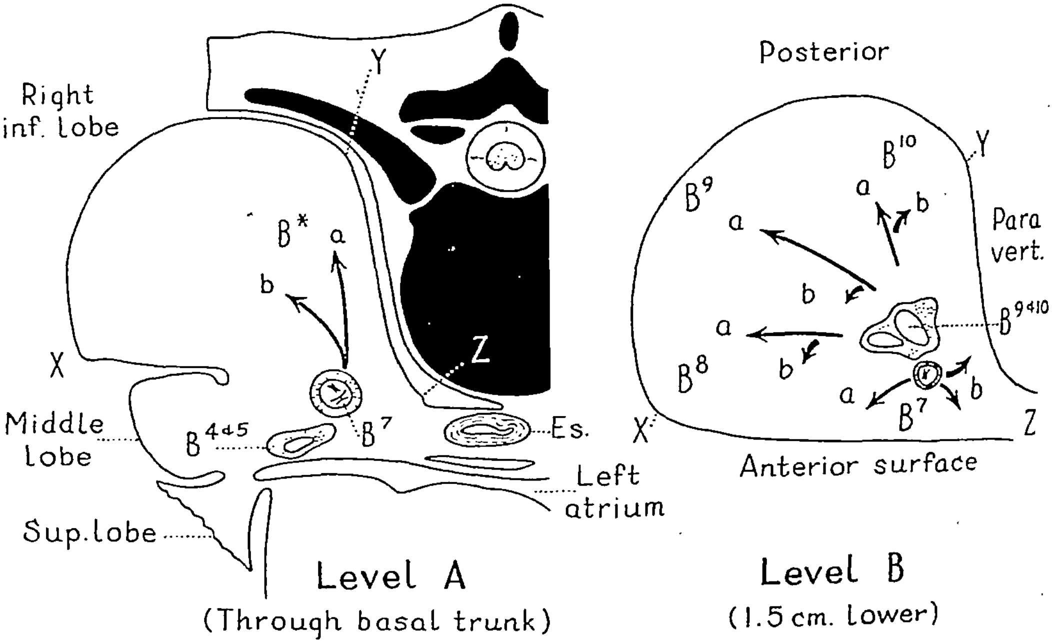

Fig. 1.—Transverse sections of right lower lobe at two levels: *Level A*, section just below orifice of superior segmental bronchus (B^6). to show origin of subsuperior bronchus (B^*) from common stem of $B^{8, 9+10}$; the branches of B^* are so lettered as to correspond to the overlying branches of B^6 (see Fig. 3). *Level B*, section just below origin of B^*, to show sectors of the lung supplied by the four basal bronchi (B^7, B^8, B^9, B^{10}. *X*, interlobar margin, separating anterior (interlobar) from costal surface of lobe; *Y*, costoparavertebral margin, separating paravertebral from costal surface of lobe; *Z*, pulmonary ligament, separating anterior from paravertebral surface. Arrows, at *Level B*, indicate direction of bronchi. The *b* branches of B^8, B^9, B^{10} are descending toward the diaphragm. Note that B^7a supplies the anterior surface, B^7b the paravertebral (and anterior) surface.

THE GROSS CONFIGURATION OF THE RIGHT LOWER LOBE

The right lower lobe somewhat resembles a cone that has been flattened anteriorly and medially, left rounded on its posterior and lateral side, and then telescoped from below. It thus presents four surfaces—a slightly curved anterior surface flattened by the upper and middle lobes, a paravertebral surface lying against the vertebral column and grooved by the esophagus, a costal surface molded by the curving ribs, and a concave diaphragmatic surface indented by the dome of the liver. As seen in cross section (Fig. 1) the anterior and costal surfaces are separated by the right-angled interlobar margin (X), the costal and vertebral surfaces by an obtuse costoparavertebral margin (Y), and the anterior and paravertebral surfaces by the hilum and pulmonary ligament (Z).

The *costo-paravertebral margin* is barely noticeable in a fresh specimen, but it has been arbitrarily indicated by a line in the figures on the right side of Plates 1 to 4 (see Plate 1 for designation), since the position of the segments cannot be defined easily without such a landmark.

The *interlobar margin* is shown in the figures on the left side of Plate 1. This is the edge which marks the posterior border of the interlobar or oblique fissure. As pointed out by Brock (1942), this fissure begins at the fifth rib or the fifth interspace (one segment lower than is usually described in the anatomic texts) and follows the sixth rib down to the diaphragm.

The *anterior* (or interlobar) *surface* is not a flat plane but is often gently curved. Superiorly, as the lobe thins out and becomes nearly semicircular in cross section, the anterior surface faces somewhat laterally. This portion is usually fused in varying degree with the posterior segment (B^2) of the upper lobe. (See Plates 1 and 4, No. 40 and 49.) Inferiorly, the anterior surface faces more medially and is characterized by a fossa which we have termed the sulcus of the *inferior* vena cava (Plate 1) to distinguish it from the fossa for the superior vena cava which carries the anatomic term, sulcus venae cavae. The former is also a substantial groove. This was recognized by Braus (1924), who gave it the vernacular German name of *Delle*.

Laterally, the inferior portion of the anterior surface is crossed by the middle lobe. The lower border of the middle lobe usually passes from the hilum toward the lower lateral angle of this anterior surface. (See line separating subsegments B^{6}b and B^{7}a in specimen No. 39, Plate 4.) The position of this inferior margin of the middle lobe is not marked by any ridge or groove on the anterior surface of the lower lobe, nor have any of the fifty specimens presented a fusion of the middle lobe along this line. However, the proximal end of the middle lobe frequently fuses to the lower lobe at the inferior or lateral margins of the hilum (see No. 40, Plate 1).

The superior margin of the middle lobe coincides with the *interfissural crest* (Plate 1) which separates the surfaces on the inferior lobe that are covered, respectively, by the upper and the middle lobe. This line usually coincides with the division between upper and lower portions of the inferior lobe, the upper half representing the distribution of the superior segmental bronchus (B^6) and the lower half the zone supplied by the two basal segmental bronchi (B^{7+8}).

THE SUPERIOR SEGMENTAL BRONCHUS (B^6)

The superior segmental bronchus is the first posterior branch of the right lower lobe stem and is situated just inferior to the level of the orifice of the middle lobe bronchus and nearly opposite to it. It has three principal rami (Fig. 2, *A*): a medial (B^{6}a), a superior (B^{6}b) and a lateral (B^{6}c).* This does

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*The medial is the paravertebral ramus of Brock (1943). It was originally designated the posterior ramus by Boyden (1945), but the additional information obtained in this study suggests that medial is more accurate than posterior. Also, in conformity to the method of numbering branches of the right lung in counterclockwise order, the letters designating these rami have been revised.

EXPLANATION OF PLATES

(Sketches rendered for publication by Lawrence B. Benson)

Sketches of injected fresh specimens of the right lower lobe of the lung, showing distribution of segmental bronchi. The large drawings on the left side display the anterior or interlobar surface; those on the right side, the costal surface as viewed posteriorly (X1⅓). The smaller figures represent the diaphragmatic surface (X1⅙).

Color scheme: *Yellow color,* segment supplied by superior bronchus (B^6); B^6a, b, c, distribution of its medial, superior, and lateral rami. *White color,* segment supplied by subsuperior bronchi (B^* and/or BX^*). *Blue color* (anterior surface), segments supplied by medial basal bronchus (B^7); a, b, distribution of anterior and medial rami. *Red color,* segment supplied by anterior basal bronchus (B^8); a, b, distribution of lateral and basal rami. *Blue color* (costal surface), segment supplied by lateral basal bronchus (B^9); a, b, distribution of lateral and basal rami. *Green color,* segment supplied by posterior basal bronchus (B^{10}); a, b, laterobasal and mediobasal rami. (The highest branch of B^{10} (BX^*) is left uncolored when it supplies the subsuperior zone.)

Plate 1.—*Specimen 43,* The superior segment (B^6) is typical as to size, angle of placement, and distribution of rami. The subsuperior segment (B^*) is typical in position (except that it is pushed somewhat laterally by the invading ramus of B^7) but it belongs in that small group (14 per cent) in which the segment is supplied only from level 2 of the stem bronchus, that is by a subsuperior proper (B^*). In this case, the bronchus arises just above the orifice of B9. The basal segments are typical except that the medial basal bronchus (B^7) is defective: although originating at its usual place, it consists of a medial ramus only (B^7b); the apparently missing anterior ramus (B^7a) is represented by an accessory branch (BX^7a) of the anterior basal, hence the latter (B^8) appears to extend atypically as far as the sulcus of the inferior vena cava.

Specimen 40, The superior segment (B^6) occupies the upper half of the lobe. Posteriorly, the lateral ramus (B^6c) predominates, and its displaced deep branch (‡) invades the subsuperior region (for normal deep branch, see Figs. 2A and 3). The subsuperior segment is thereby displaced somewhat laterally. It is supplied by the high dorsal branch (BX^*) of the posterior basal (B^{10}). The basal segments are atypical in that B^9 is absent as such, being represented by BX^9b, an accessory ramus of E^8b. Consequently in this specimen, the anterior basal segment extends more posteriorly than usual. B^7b fails to reach the diaphragm.

Plate 2.—*Specimen 48,* The superior segment (B^6) occupies an upper wedge-shaped zone of the lobe. Both it and the posterior basal segment (B^{10}) are shortened by the unusual paravertebral extent of the subsuperior segment. The latter is supplied by three bronchi—one subsuperior proper (B^*) which arises just beneath the orifice of B^7, and two accessory subsuperiors (BX^*) which arise several centimeters lower down from B^{10}a and B^{10}b. The lateral basal segment (B^9) is shortened by the unusual spread at B^8a.

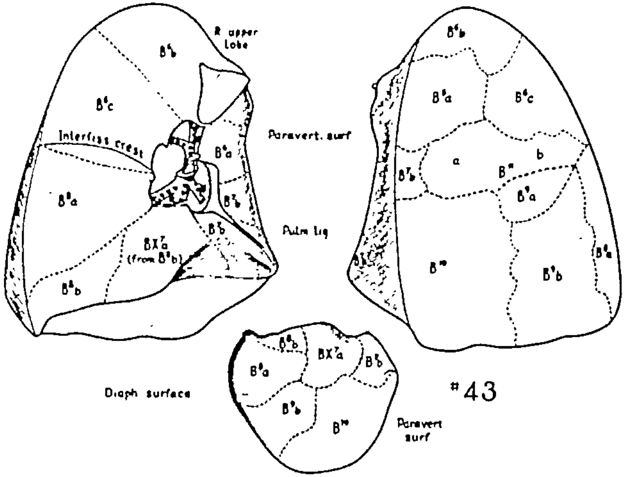
Specimen 42, The superior segment (B^6) occupies an oblique half or less of the lobe. Again ramus B^6c predominates posteriorly. The subsuperior segment is a small mid-posterior zone supplied by BX^* (10). The medial basal segment (B^7) is demarcated by a deep fissure which extends to the center of the diaphragmatic surface. The anterior basal (B^8) is anomalous in that while its bronchus arises from the usual position it consists of the lateral ramus only (B^8a). (This, in turn, has an accessory branch [BX^6c] which takes over part of the territory above the interfissural crest, that is, the zone usually supplied by the superior bronchus, B^6.) The basal portion of the anterior basal segment (BX^8b) is taken over by an accessory branch of the lateral basal bronchus (B^9).

Plate 3.—*Specimen 50,* The superior segment (B^6) is obliquely placed. In contrast to that of Specimen 40 (Plate 1) the segment in this specimen descends further anteriorly than posteriorly. Also B^6c predominates posteriorly. The subsuperior segment is supplied by two subsuperiors proper, the first B^* arising midway between the orifices of B^8 and B^9, the second B^* just above the orifice of B^9, yet, curiously enough, they terminate side by side. Because of the low origin of the second B^*, the accessory subsuperior (BX^*) was presumably forced to distribute much lower than usual; for this reason it has not been included in the subsuperior segment although its designation has been retained. This is also the only specimen encountered in which the lateral branch (B^9a) of the lateral basal segment reached the diaphragm. Otherwise, the basal segments are typical.

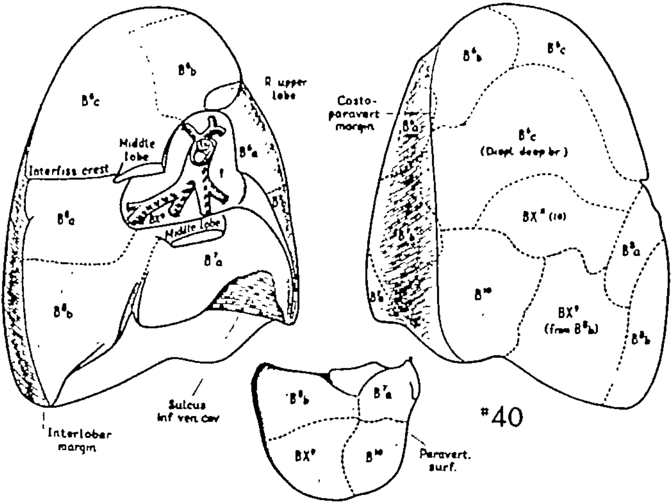
Specimen 46, This and No. 42 (Plate 2) illustrate that 38 per cent of specimens in which the subsuperior segment is supplied only by the BX^* ramus of B^{10}. It differs from that of No. 42 in that it extends into the paravertebral area. The other segments are typical.

Plate 4.—*Specimen 39,* This is the only specimen encountered in which there were two subsuperior bronchi arising exclusively from the stem of the posterior basal (B^{10}). Only the upper one, however, may properly be considered as supplying the subsuperior segment. Also, *this specimen resembles No. 42 (Plate 2) in that* B^8a takes over a portion of the superior segment by virture of its accessory ramus, BX^6c. It differs in that B^8b is normal in origin and not a branch of B^9 as in No. 42.

Specimen 49, This is the only specimen encountered in which the anterior basal segment (B^8) existed as a separate lobe. As shown in diaphragmatic view, the segment consisted of a cleft lobe which seemed to have protruded from its position between the medial basal (B^7) and lateral basal segment (B^9). This specimen is also the only one encountered which had a distinct cleft between superior and basal segments. It is also peculiar in the extent to which the superior segment is fused to the upper lobe.

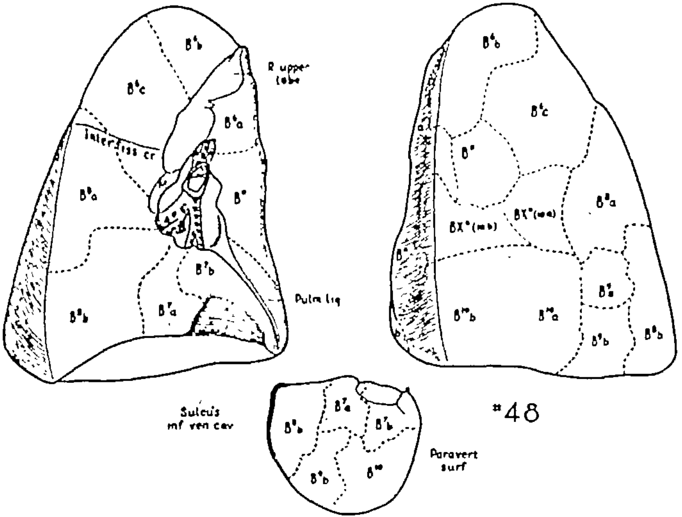

R upper lobe
$B^{6}b$
$B^{6}c$
Interfiss crest
$B^{6}a$
Paravert. surf
$B^{8}a$
$B^{7}b$
Pulm lig
$BX^{7}a$ (from $B^{8}b$)
$B^{8}b$
$B^{6}b$
$B^{6}a$
$B^{6}c$
$B^{7}b$
a
b
B^{*}
$B^{9}a$
$B^{8}a$
B^{10}
$B^{9}b$
$B^{8}b$
$BX^{7}a$
$B^{7}b$
$B^{8}a$
Diaph surface
$B^{9}b$
B^{10}
Paravert surf

#43

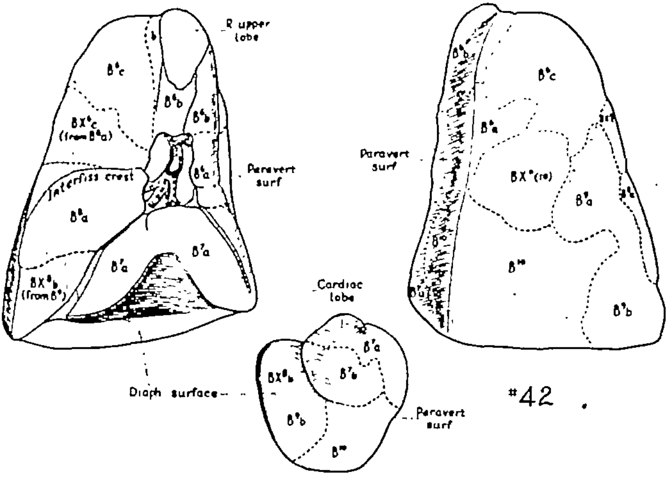

$B^{6}b$
R upper lobe
$B^{6}c$
Middle lobe
$B^{6}a$
Interfiss crest
$B^{8}a$
$B^{7}b$
BX^{9}
Middle lobe
$B^{7}a$
$B^{8}b$
Sulcus inf ven cav
Interlober margin
$B^{6}b$
$B^{6}c$
Costo-paravert margin
$B^{6}a$
$B^{6}c$ (Diaph deep br)
BX^{*} (10)
$B^{8}a$
B^{10}
BX^{9} (from $B^{8}b$)
$B^{8}b$
$B^{8}b$
$B^{7}a$
BX^{9}
B^{10}
Paravert. surf.

#40

Plate 1.

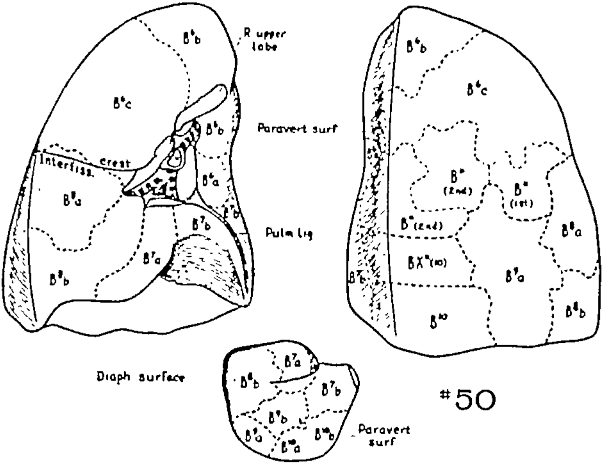

B^6b
R upper lobe
B^6c
B^6a
Interfiss cr
B^8a
B^*
B^7b
Pulm lig
B^8b
B^7a
B^6b
B^6c
B^*
B^8a
BX*(mb)
BX*(wa)
B^9a
$B^{10}b$
$B^{10}a$
B^9b
B^8b
Sulcus inf ven cav
B^7a
B^8b
B^7b
Paravert surf
B^9b
B^{10}

#48

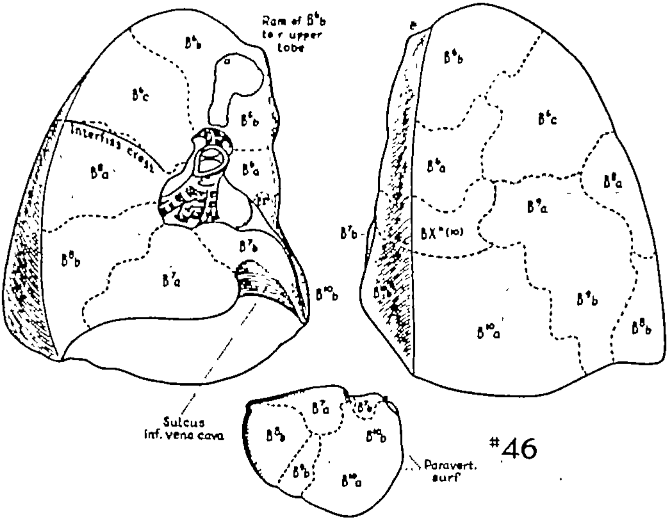

R upper lobe
B^6c
B^6b
BX^6c (from B^8a)
Paravert surf
Interfiss crest
B^6a
B^8a
B^7a
B^7a
BX^8b (from B^9)
Diaph surface
Cardiac lobe
B^7a
B^7b
BX^8b
B^9b
B^{10}
Paravert surf
B^6c
B^6a
$BX^*(re)$
B^9a
B^{10}
B^9b
Paravert surf

#42

Plate 2.


B^6b
R upper lobe
B^6c
B^6b
Paravert surf
Interfiss. crest
B^6a
B^8a
B^7b
Pulm lig
B^7a
B^8b
B^6b
B^6c
B^* (2nd)
B^* (1st)
B^*(2nd)
B^8a
BX^*(10)
B^9a
B^7b
B^{10}
B^8b
Diaph surface
B^7a
B^8b
B^7b
B^9b
B^9a
$B^{10}b$
$B^{10}a$
Paravert surf

#50


Ram of B^6b to r upper lobe
B^6b
a
B^6c
B^6b
Interfiss crest
B^8a
B^6a
B^8b
B^7b
B^7a
$B^{10}b$
Sulcus inf. vena cava
B^6b
B^6c
B^6a
B^8a
B^9a
BX^*(10)
B^7b
$B^{10}b$
B^9b
$B^{10}a$
B^8b
B^7a
B^7b
B^8b
$B^{10}b$
B^9b
$B^{10}a$
Paravert. surf

#46

Plate 3.

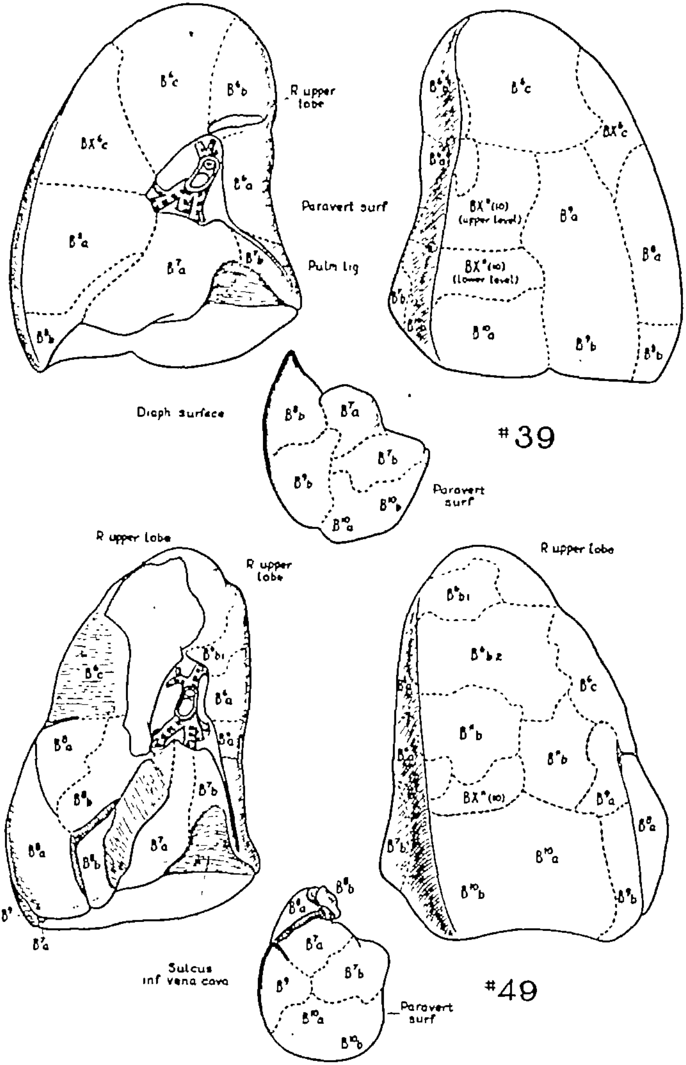

$B^{6}c$
$B^{6}b$
R upper lobe
$BX^{6}c$
$B^{6}a$
Paravert surf
$B^{8}a$
$B^{7}a$
$B^{7}b$
Pulm lig
$B^{8}b$
$B^{6}b$
$B^{6}c$
$B^{6}a$
$BX^{6}c$
$BX^{9}(10)$ (upper level)
$B^{9}a$
$B^{8}a$
$BX^{9}(10)$ (lower level)
$B^{7}b$
$B^{10}b$
$B^{10}a$
$B^{9}b$
$B^{8}b$
Diaph surface
$B^{8}b$
$B^{7}a$
$B^{7}b$
$B^{9}b$
$B^{10}b$
$B^{10}a$
Paravert surf
#39
R upper lobe
R upper lobe
$B^{6}b1$
$B^{6}c$
$B^{6}a$
$B^{6}a$
$B^{8}a$
$B^{8}b$
$B^{7}b$
$B^{8}a$
$B^{8}b$
$B^{7}a$
B^{9}
$B^{7}a$
R upper lobe
$B^{6}b1$
$B^{6}b2$
$B^{6}c$
$B^{6}b$
$B^{6}a$
$B^{9}b$
$BX^{9}(10)$
$B^{9}a$
$B^{8}a$
$B^{7}b$
$B^{10}a$
$B^{10}b$
$B^{9}b$
$B^{8}b$
$B^{8}a$
$B^{7}a$
$B^{7}b$
B^{9}
$B^{10}a$
$B^{10}b$
Sulcus inf vena cava
Paravert surf
#49

Plate 4.

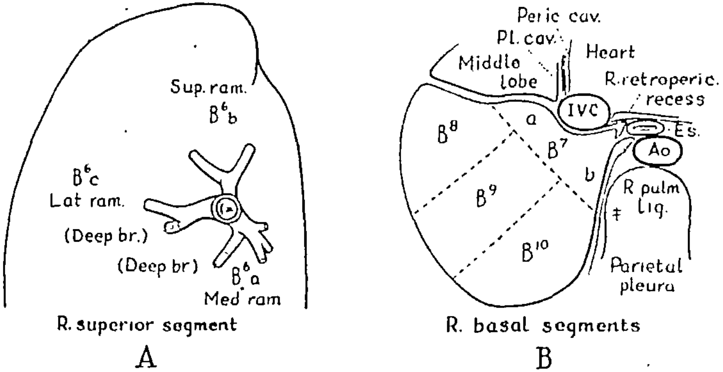


Fig. 2.—*A*, Anterior view of superior segmental bronchus (B^6), showing arrangement of its three rami—B^6a, B^6b, B^6c—in an embalmed specimen (see Fig. 7, *B*). While this represents the common type of bifurcation into B^6a + b and B^6c it is atypical in that it resembles a trifurcation. For more typical mode of branching, see B^6, Fig. 3, *C*.

B, Diagram of transverse section of the right lower lobe through the lower part of the basal segments. Note that the medial basal segment (B^7) is placed athwart the other basal segments (B^8, B^9, B^{10}) and that the latter exhibit a layering, B^8 overlapping B^9, and B^9 overlapping B^{10}. Also note that B^7 lies on both sides of the pulmonary ligament, B^7a typically extending as far as the lateral third of the anterior surface; B^7b reaching well down on the paravertebral surface (‡).

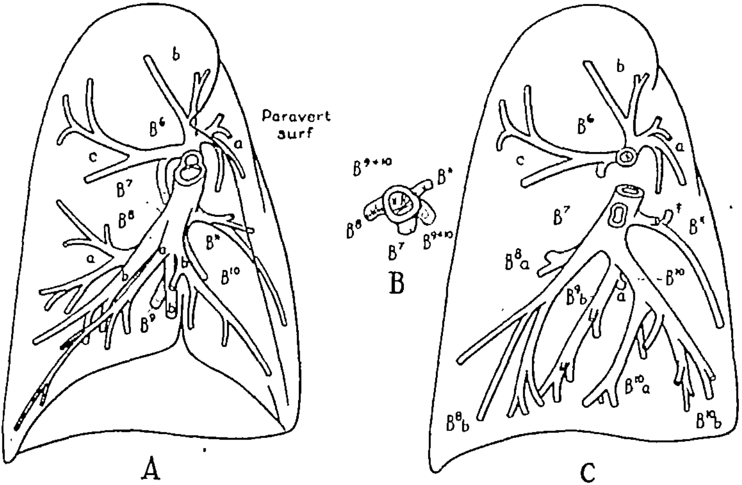


Fig. 3.—Dissection of embalmed right lower lobe as seen from anterior surface.

A, Superficial dissection of bronchi. This specimen is typical in respect to the level of origin of the segmental bronchi B^*, arising from the common stem of B^8, B^9, and B^{10}, but below B^7. It is atypical in that the subsuperior bronchus (B^*) is directed medially instead of posteriorly. For this reason, presumably, the medial ramus of the medial basal (B^7b) does not distribute to the paravertebral surface as it usually does. Correspondingly, the anterior ramus (B^7a) supplies the whole of the lower anterior surface, instead of the usual two-thirds of it. Finally, the deep branch of B^6c takes over part of the posterior distribution of B^*.

B, Orifice of basal stem just below level of B^*.

C, Deeper view of dissection following removal of B^7, the medial basal bronchus. Note deep posterior branch of the subsuperior (‡) that was hidden in *A*. Note absence of BX* (10)—the high posterior branch of B^{10} which occurs in 86 per cent of specimens. B^6, superior segmental bronchus; B^*, subsuperior bronchus; B^7, medial basal; B^8, anterior basal; B^9; lateral basal; B^{10}, posterior basal segmental bronchus.

not mean that B^6 is typically trifurcate, as implied by Brock (and suggested by the specimen drawn in Fig. 2): for the prevailing pattern (86 per cent) is a bifurcation, B^6c and $B^6a + b$; see Fig. 3 and Plate 2. (See also Table I, item 1.) The remaining items in the table demonstrate how rarely this pattern is modified. In only a few instances (10 per cent), is the segment supplied by an accessory branch of B^8 (see BX^6c, Plate 2, No. 42; Plate 4, No. 39), or by an accessory branch of the subsuperior bronchus, or directly by a branch from the basal stem (see footnotes, Table I).

TABLE I. THE SUPERIOR SEGMENTAL BRONCHUS (B^6)

1. *Bifurcates* into:			94%
1.* $B^6a + b$ and B^6c (Plate 2, No. 42)		86%	
2. Bizarre types		8%	
(1) B^6c2 and $[B^6a + c1 + b]$	2%		
(2) B^6c2 and $[(B^6a + b) + c1]$	2%		
(3)† B^6a and B^6b	2%		
(4)‡ B^6b1 and B^6b2	2%		
II. *Trifurcates* into B^6a, B^6b, B^6c			6%

*In three specimens, part of B^6c arose from the basal stem (No. 17) or from B^8; see No. 42 (Plate 2) and No. 39 (Plate 4).

†B^6c arises as BX^6c from the basal stem.

‡B^6a arises as BX^6a from the subsuperior bronchus (B^*).

B^6c1 arises as BX^6c1 from B^*; B^6c2 from basal stem.

The amount of the lower lobe tissue that is aerated by B^6 is of considerable importance to the clinician since this is frequently involved in lung abscess and frequently separated from the basal segments. For these reasons it has seemed desirable to summarize the limits of the superior segments on all three surfaces of the lower lobe (Fig. 4). Row 1 shows the extent of this segment on the anterior surface, together with the percentages in which it occupies one-third, one-half, or two-thirds or more of the surface. Rows 2 and 3 supply similar data for the costal and paravertebral surfaces, respectively. However, these figures, suggestive as they are, do not in themselves yield the most significant information. This has been obtained by ascertaining the extent of this segment in each specimen and then finding into what groups these specimens can be arranged.

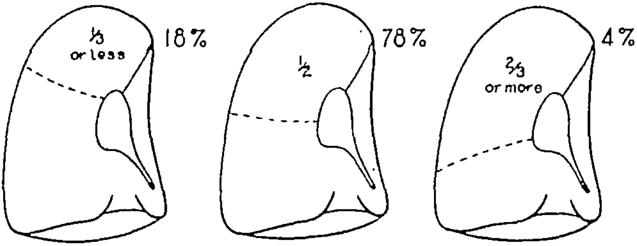
Two major patterns emerge from this analysis. In two-fifths of the specimens (38 per cent) the superior segment is obliquely fitted to the basal segments of the lower lobe. In the remainder (62 per cent), it caps the basal segments horizontally, according to conventional concepts. The whole group may be further subdivided according to whether the segment occupies the upper half

TABLE II. POSITION AND EXTENT OF THE SUPERIOR SEGMENT

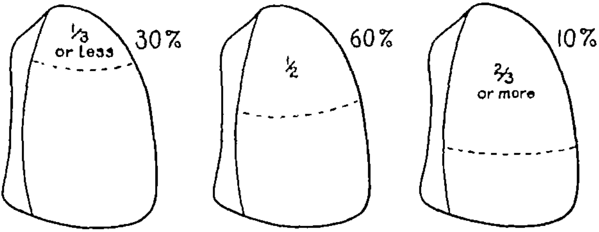
Horizontal group		62%
Upper third	8%	
Upper half	52%	
Upper two-thirds	2%	
Oblique group		38%
Upper third	6%	
Upper half	26%	
Upper two-thirds	6%	

(78 per cent), the upper third (14 per cent), or two-thirds (8 per cent) of the lobe. Details are given in Table II.

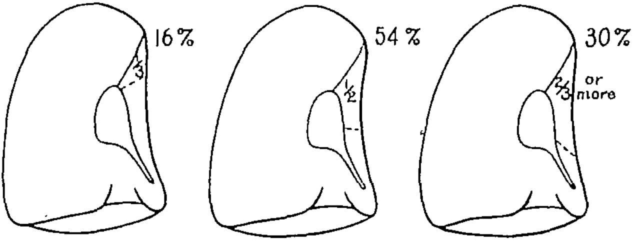
In no instance was the superior segment cut off from the basal segments by a substantial incisure, as described by Dévé (1900) in 6 per cent of 180 infant lungs—the nearest to this condition being shown in Plate 4 (No. 49)—nor was it



Anterior Surface



Costal Surface

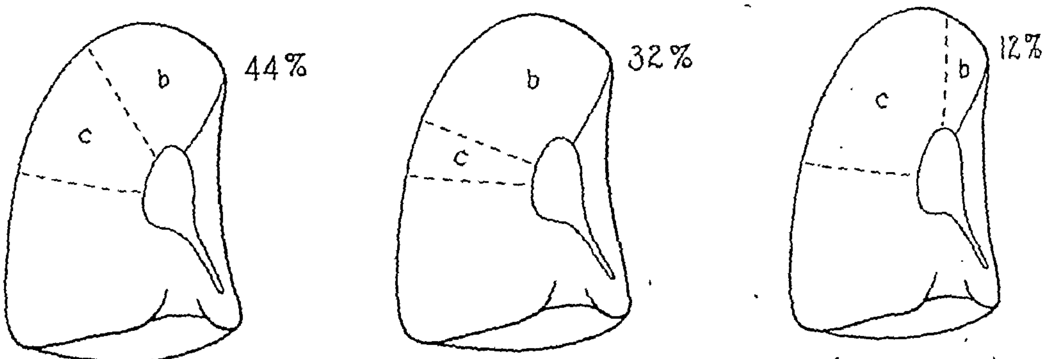


Paravertebral Surface

Fig. 4.—Diagrams illustrating varying extent of the superior segment on anterior, costal, and paravertebral surfaces. Percentages indicate frequency of given distributions.

ever associated with the 36 per cent of incisures demarcating the medial basal segment in our series. Dévé observed a coexistence of these incisures in 11 per cent of infant lungs. (This figure includes incomplete fissures.)

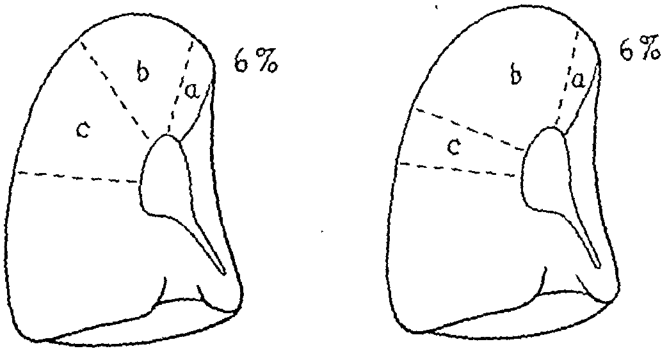
The composition of the anterior and costal surfaces of the segment is displayed in Figs. 5 and 6, respectively. In the prevailing pattern (Fig. 5, 44 per cent), the anterior surface is supplied exclusively and equally by B^6b and B^6c. On the costal surface (Fig. 6), the two most common patterns are those in which rami b and c again divide the area (34 per cent), and those in which rami a, b, and c participate equally (30 per cent). On the paravertebral surface, which has not been plotted because of its constancy, B^6a is present and predominant in 98 per cent of cases. B^6b is the only other ramus which invades this area even to a minor degree and then only in 44 per cent of specimens.



B^6c and B^6b equal (B^6a absent)

B^6b predominant (B^6a absent)

B^6c predominant (B^6a absent)



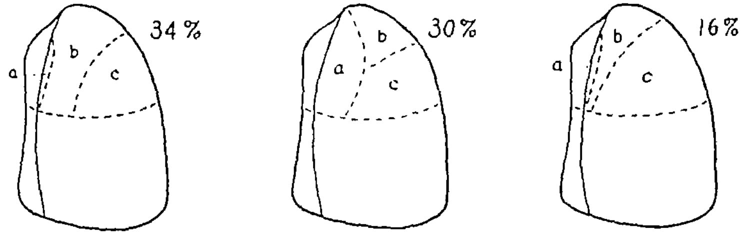
B^6c predominant

B^6b predominant

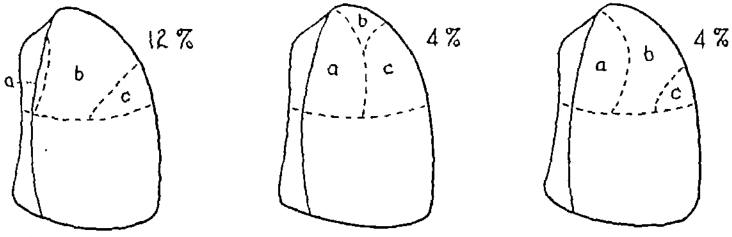
Fig. 5.—Diagrams illustrating varying extent of anterior surface of the superior segment supplied by individual rami of B^6.

Of the three rami just described, the *medial* (B^6a) is most worthy of attention since this is the one which descends furthest. This agrees with Brock's observation that the downward direction of this branch may be so increased that it runs almost vertically. Thus in 54 per cent of specimens (Fig. 4), it supplies one-half of the paravertebral surface (with or without a small portion of B^6b) and in another 30 per cent it covers two-thirds or more of the surface. Indeed, in two instances it almost reaches the diaphragm (see Fig. 9). From the standpoint of resection of the segment, the importance of this cannot be overemphasized. Another point of interest is that in 38 per cent it develops a deep branch (Fig. 2, *A*) which takes over a substantial portion of the costal surface (see Plate 3, No. 46), but it was never found to predominate on that surface.

Next in interest is the *lateral ramus* (B^6c). In 84 per cent this has a deep posterior branch (Fig. 3, *A*) and occasionally (16 per cent) it may take over most of the costal surface of the segment (Plates 2 and 4, No. 42 and No. 39). In one instance, what is presumably to be interpreted as this deep branch arose coordinately with the common stem of $B^6a + b$ (see orifice ‡, Plate 1, left side, No. 40). Evidence for this may be found in Plate 3 (No. 50) where the typically placed deep branch of B^6c supplies a comparable zone.



B^6b and B^6c predominant B^6a, B^6b, B^6c equal B^6c predominant



B^6b predominant B^6a and B^6c predominant B^6a and B^6b predominant

FIG. 6.—Diagrams illustrating varying extent of costal surface of the superior segment supplied by individual rami of B^6.

The *superior ramus* (B^6b) is distributed to both sides of the upper portion of the lobe.

From the standpoint of resection, the common stem of the three rami of B^6 is long enough to permit ligation in 92 per cent of specimens. The same is true of the common stem of $B^6a + b$ in 88 per cent and of the lateral ramus (B^6c) in 100 per cent of specimens.

In concluding discussion of the superior segment attention is again called to the fact that on the right side the upper lobe is usually fused to a greater or lesser degree with the anterior surface of the superior segment where that plane curves to join the paravertebral surface. Usually this area is contiguous to B^6b. In this region (see Plate 3, No. 46), a branch of B^6b was observed to supply the parenchyma of the right upper lobe in five out of eleven consecutive specimens examined for this purpose. A similar situation was noted by Bell (1941). In a personal communication he stated that this fact was observed in a case of lobar pneumonia in which the consolidation extended from the right lower lobe to the adjoining area of the right upper lobe. Post-mortem dissection showed that the bronchial twig from the superior segmental bronchus supplied this area.

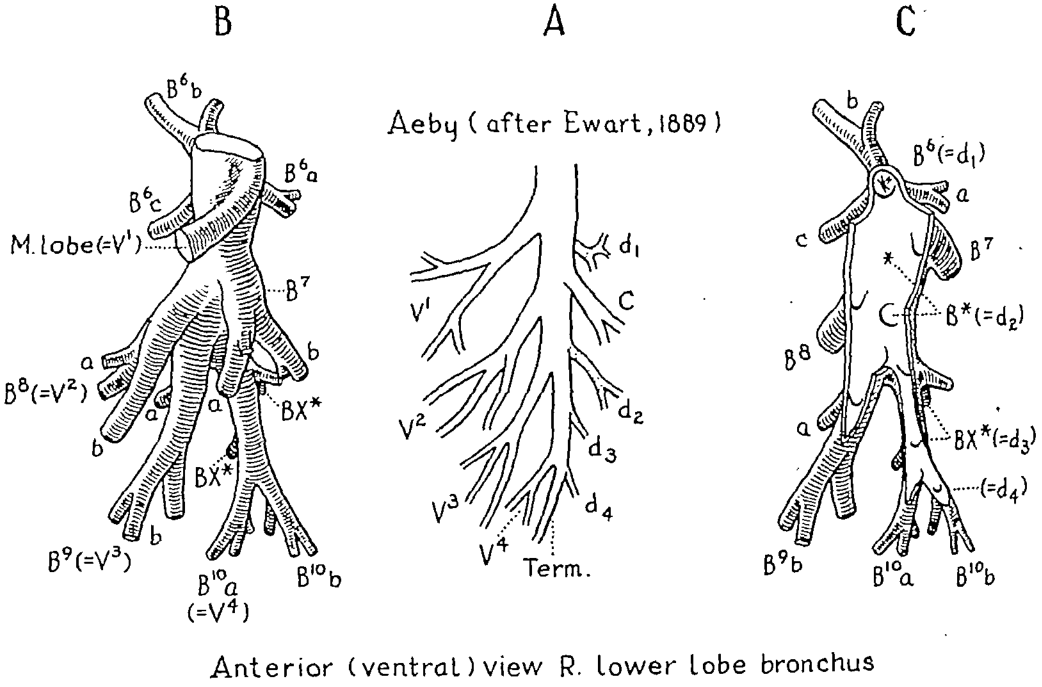


Fig. 7.—Sketches showing the origin and relations of posterior (dorsal) branches of *the right lower lobe bronchus.*

A, Aeby's diagram illustrating "monopodic" branching of a stem bronchus into a series of ventral (V^{1-4}) and dorsal d_{1-4}) rami. *C*, cardiac bronchus, which does not fall into either of the two series. *Term.*, termination of "stem bronchus."

B, Dissection of embalmed specimen (No. 12, 816), for comparison with Aeby's diagram.

C, Same specimen as in *B*, with anterior wall of bronchial tree removed to show orifices of dorsal rami. In this particular specimen, five instead of Aeby's four dorsal rami occurred. (The first dorsal is represented by B^6.) A sixth is frequently found in certain other specimens at the point indicated by an asterisk, that is on the common stem of B.$^{8, 9+10}$ Considering the six in order from above downward. No. 2 and 3 are considered as equivalent to Aeby's d_2. Dorsal bronchi on this part of the stem are here designated the subsuperior proper (B^*), No. 4 and 5 (on the stem of B^{10}) are considered as equivalent to Aeby's d_3 and are here designated as the accessory subsuperior, BX^* (10). Bronchi arising in positions 2 to 5 usually supply the subsuperior segment, and not successively lower zones, as might be inferred from their position on the stem bronchus.

THE SUBSUPERIOR BRONCHUS (B^*)

The subsuperior bronchus, as indicated by its name, arises beneath the superior bronchus (B^6) either from the posterior portion of the basal trunk (B^*, Fig. 7, *C*) or from the upper portion of the posterior basal bronchus (BX^*, Fig. 7, *C*). It is distributed to the middle level of the posterior costal surface of the lobe. It may extend onto the paravertebral or onto the lateral costal surface. This is one of the zones which has been considered to be most vulnerable to aspirated material (Neil, Gilmour, and Gwynne, 1937).†

The first to recognize a series of dorsal or posterior branches on the stem bronchus of man was the Swiss comparative anatomist, Aeby (1880). He postulated a monopodic branching consisting of four ventral and four dorsal rami. In this he was guided by the prevailing mammalian pattern in which the thorax is long and narrow and the heart placed near the middle of the thorax. Such a scheme is shown at *A* in Fig. 7. Ewart (1889), however, took exception to this rationalization, stating that due to the different shape of the human thorax, which is compressed from front to back, with the heart placed inferiorly, the branching of the human bronchial tree is of necessity more radial, and more dichotomous, than monopodic (see *B*, Fig. 7). Furthermore it has been our experience that the four dorsal branches postulated by Aeby do not occur regularly in the right lower lobe bronchus of man, and that potentially there are six sites, as shown at *C* in Fig. 7: (1) the site of the first dorsal branch of Aeby (d_1), which is the superior segmental bronchus (B^6); (2) the dorsal branch between B^7 and B^8 (marked by an asterisk in *C*, Fig. 7); (3) the dorsal branch between B^8 and $B^{9,10}$ (Aeby's d_2); (4) the dorsal branch at the upper end of B^{10}; (5) the dorsal branch at the middle of B^{10} (Aeby's d_3); and (6) the dorsal branch of $B^{10}b$ (Aeby's d_4). However, for the sake of simplification, dorsal branches 2 and 3 are here grouped together and considered as corresponding to Aeby's d_2, and dorsal branches 4 and 5 as corresponding to Aeby's d_3. It is thus convenient to refer to the subsuperior bronchus as arising either at level d_2 or level d_3, that is, from some portion of the main stem or from B^{10}, respectively. The first will be referred to as the *subsuperior proper*, with the designation B^* (after Boyden, 1945) and the second as the *accessory subsuperior*, with the designation BX^*.

In considering that both B^* and BX^* represent a subsuperior (in which view Brock also concurs) we have been guided by the fact that in the absence of a B^* the BX^* distributes to the subsuperior zone rather than to a distinctly lower level, a fact which again may be referred to the radial type of branching of the human lung.

Somewhat similar to Fig. 7, *C* is a sketch presented by Neil, Gilmour, and Gwynne (1939), showing the opened bronchial tree of a 4-year-old child in which three subsuperiors are present (their Fig. B). The first two correspond to the d_2 level, and the third—if the specimen had had the usual high bifurcation of B^{9+10}—to the d_3 level, as postulated in Fig. 7, *C*. In general, these authors stated that there are one to three present, and this corresponds to our observations; for inspection of Table III (Item I) will show that one or more subsuperiors are present in every specimen. In 50 per cent only one occurs (Item II:1); in 34 per cent two are present (Item II:2); in 14 per cent three occur (Item II:3), and in one specimen even four such dorsal rami were noted (Item II:4).†

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†At first unaware of Ewart's earlier identification of it (as the "lesser posterior horizontal") they claimed discovery of a new segment—naming it the "subapical"; and, indeed, it had been overlooked by Kramer and Glass (1932). Neil's excellent description of it, with emphasis on its clinical importance is as follows:

> "The posterior region of the lower lobe is supplied, according to all previous publications to which we have had access, by the apical bronchus [B^6] in the upper portion and the paravertebral [B^{10}] in the lower. We are convinced that an intermediate separate segment exists and can be demonstrated, and that it is the site of localized pathological change, as shown by radiology and bronchoscopy. It is an anatomical gap in the distribution between the apical and paravertebral bronchi in all illustrations we have seen. We have found that it is supplied by these subapical bronchi [B^* and BX^*]. This has been shown by inflation in specimens and by lipiodol fillings in patients."

TABLE III. SUBSUPERIOR BRONCHI (B^* AND BX^*)

I. Frequency of occurrence in 50 specimens			
1. Arises exclusively from level d_2 (as branch of main lower lobe bronchus between the superior and the basal segmental bronchi)			14%
2. Arises exclusively from level d_3 as an accessory subsuperior, $BX^*(10)$, i.e., one or more from the posterior basal bronchus (B^{10})			38%
3. Arises from both levels in same specimen			48%
			100%
II. Detailed analysis			
1. Subsuperior represented by single bronchus			50%
(1) Arising at d_2 level (as B^* proper)		14%	
(a) From common stem of $B^{8, 9, 10}$	6%		
(b) From common stem of $B^{9, 10}$	8%		
(2) Arising at d_3 level, as $BX^*(10)$		36%	
2. Subsuperior represented by two bronchi			34%
(1) One from level d_2 (B^*), and one from level d_3, as $BX^*(10)$		32%	
(2) Two from level d_3, as $BX^*(10)$		2%	
3. Subsuperior represented by three bronchi			14%
(1) Two from level d_2 (B^*) and one from level d_3, as $BX^*(10)$		10%	
(2) Two from level d_3, as $BX^*(10)$ and one from level d_2, as B^*		4%	
4. Subsuperior represented by four bronchi (Two from each level)			2%
			100%
III. Origin of B^* (at d_2 level). (Items I:1 and I:3)			62%
1. From basal stem (level of B^7)		12%	
2. From common trunk of $B^{8, 9, 10}$		22%	
3. From common trunk of $B^{9, 10}$		14%	
4. Two: one from common trunk of $B^{8, 9, 10}$ and one from $B^{9, 10}$		6%	
5. Two from common trunk of $B^{9, 10}$		8%	

At this point one may ask why Neil, Gilmour, and Gwynne have not been followed in giving the subsuperior the status of a segmental bronchus. The answer lies in their observations and ours, namely, that the number and level of origin of bronchi supplying this middle posterior zone are variable, hence it is less confusing to give it a designation (B^*) that is noncommittal as to numerical sequence.

Another point made by Neil and his co-workers is that they have observed the subsuperior (subapical) "arising from the dorsal, mesial, or lateral aspect of the right main bronchus." Similarly, we have found that occasionally B^* inclines to the medial or lateral side. In the former position it may give rise to the medial ramus of the medial basal bronchus (B^7b)‡; but in general

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†It should be pointed out that the precise level of branching of the bronchi can be determined only by opening the stem bronchus. That is, one cannot always tell from the external view whether a dorsal ramus is originating from the upper part of B^{10} or the lower portion of B^{9+10}. This emphasizes the necessity of preoperative *bronchoscopy* or bronchography.

‡In Fig. 3A, B^* is medial and is taking over part of the territory of B^7, but has not replaced it.

Brock's description applies to the usual arrangement, namely that "typically [the subapical] arises from the posterior wall of the lower lobe bronchus—as it were, in series with [the apical bronchus]—. When it does not arise from the lower lobe stem it may originate from the posterior basal bronchus—still roughly parallel to [the apical bronchus]." Using Brock's definition, however, we have found the subsuperior occurring in 100 per cent of our smaller series of dissected specimens, in contrast to the 48 per cent incidence in his larger series of 170 bronchograms.

Hitherto the subsuperior has been discussed from the standpoint of its origin on the bronchial tree. Equally important is its distribution on the surface. Turning first to the subsuperior proper (B^*), analysis of the fifty injected specimens shows that in all but one of the thirty-one specimens in which it occurs (Table IV), it aerates at least the posterior sector of the right lower lobe. In 36 per cent it distributes to at least the posterior and the adjacent paravertebral area, and in 32 per cent it aerates at the least the posterior and the adjacent posterolateral area. It would seem, therefore, that to be a subsuperior proper this bronchus must supply at least the posterior sector but that it may spread either medially (B^*, Plate 2, No. 48) or laterally (B^*, Plate 1, No. 43) or in both directions (B^*, Plate 4, No. 49).

For percentages of types see Table IV.

TABLE IV. DISTRIBUTION OF THE SUBSUPERIOR (B^*)

AREA	PER CENT
Posterior and adjacent paravertebral	20
Posterior and adjacent posterolateral	16
Posterior, paravertebral, and posterolateral	16
Posterior alone	8
Posterolateral alone	2

When the subsuperior proper is absent, its zone is usually supplied by a dorsal ramus arising from the posterior basal bronchus (B^{10})—level d_3 of Aeby. This is the ramus we have called the accessory subsuperior, BX^* (10). It, too, almost always supplies at least the posterior sector (Plate 2, No. 42) but may spread medially to the paravertebral area (Plate 3, No. 46) or laterally to the posterolateral zone; or it may be displaced medially or laterally (Plate 1, No. 40) by one of the deep branches of B^*. For percentage of types, see Table V.

TABLE V. DISTRIBUTION OF THE ACCESSORY SUBSUPERIOR (BX^*) IN THE ABSENCE OF A SUBSUPERIOR (B^*)

AREA	PER CENT
Posterior alone	28
Posterior and adjacent paravertebral	4
Posterior and adjacent posterolateral	4
Posterolateral alone	2
Total	38

When both the subsuperior proper and the accessory subsuperior are present (48 per cent), the latter either distributes to the area beneath the former (BX^*, Fig. 9) or shifts medially, or to the lateral side of it (BX^*, Plate 2, No. 48), or telescopes it (Fig. 8). In only one-sixth of these specimens does BX^*(10)

supply so low a zone as to be considered part of the posterior basal rather than the subsuperior segment (namely, No. 50, Plate 3).†

When there are two accessory subsuperiors (8 per cent), one may lie below the other, in the posterior sector, or they may lie side by side (BX*, Plate 2, No. 48). If the lower one distributes too inferiorly it may be considered a part of the posterior basal segment (namely, No. 39, Plate 4).†

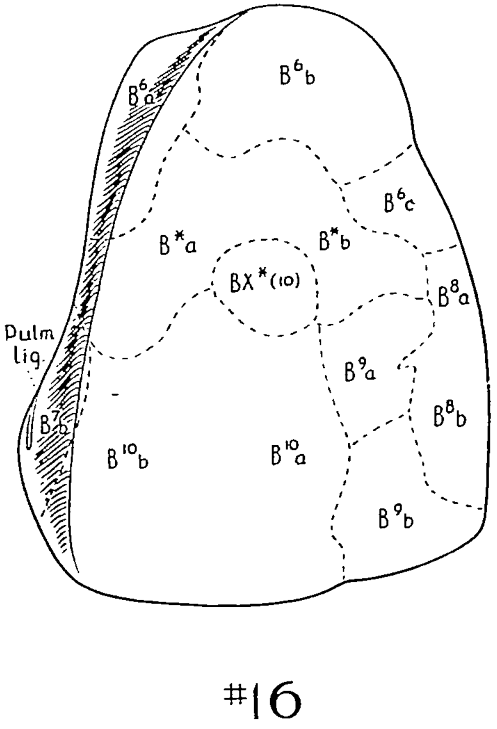

Fig. 8.

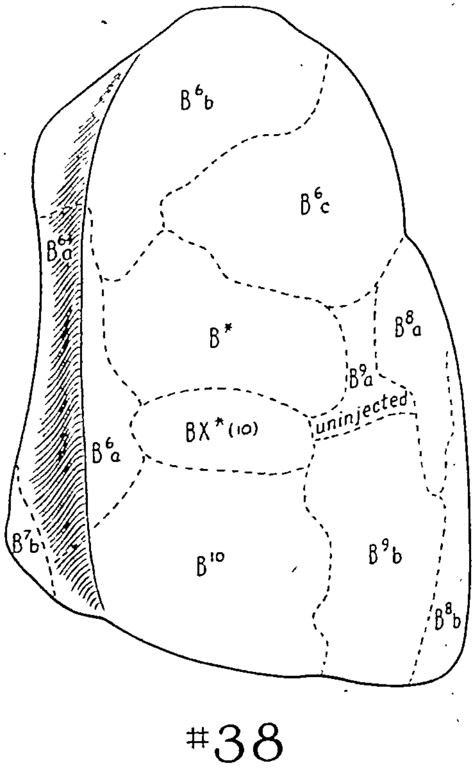

Fig. 9.

Figs. 8 and 9.—The costal surface of two injected specimens showing varying distributions of the subsuperior proper (B*) and of the accessory subsuperior bronchus (BX*). In each specimen B* arises at a level between B^7 and B^8, and BX* several centimeters below this from B^{10}, but in No. 16, BX* telescopes the zone supplied by B*, while in No. 38, it lies inferior to it. These specimens belong to that 48 per cent of lungs in which the subsuperior proper and the accessory subsuperior are present in the same lobe.

Specimen No. 38 also illustrates the fact that B^{6}a, the medial ramus of the superior segment, may take over almost the whole of the paravertebral area. In other words the superior segment may extend nearly to the diaphragmatic surface.

Of special interest are the 12 per cent of specimens in which two subsuperiors are present in addition to one, or even two, accessory subsuperiors (Table III, Item II:3[1] and II:4). In the former type, one B* may arise above the other but they distribute to zones lying side by side, as B* (first) and B* (second) in Plate 3, No. 50. Thus, they simulate the zone supplied by the two branches of a single subsuperior (B,* a and* b, Plate 1, No. 43; see also Fig. 1,*A*).

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†In such specimens the bronchi which by origin were destined to be accessory subsuperiors have been deflected downward by other bronchi. In these few specimens one thus encounters a transitional pattern of Nature which confounds man-made classifications. We have chosen to meet the dilemma by retaining the number BX*, to indicate that the bronchus has the same origin as that which supplies the subsuperior zone, but giving the zone the color of the posterior basal segment (see Plate 3, No. 50).

THE BASAL BRONCHI (B^{7-10})

The basal bronchi are four in number and arise from the basal trunk—that portion of the lower lobe bronchus which is distal to the superior segmental bronchus (B^6). As seen in cross section (Fig. 1,*B*), the major branches of the basal stem are seen to radiate in different directions. The *medial basal* (B^7), the highest branch, arises anteriorly (Fig. 3,*A*) and is distributed to the anterior and paravertebral surfaces (Fig. 1). The next branch, the *anterior basal* (B^8), arises anterolaterally (Fig. 3,*C*) and distributes to the anterior surface and lateral costal area. The next two segmental bronchi arise from a common stem which passes downward in a somewhat posteromedial direction (Fig. 3,*C*). The *posterior basal* (B^{10}) continues the direction of the common stem and distributes to the paravertebral surface and posterior costal surface. The *lateral basal* (B^9) diverges in a posterolateral direction, supplying the posterolateral sector of the costal surface. For this reason it appears to be a lateral branch (V_2) of the stem bronchus of Aeby (Fig. 7,*A*). It is useful to think of it this way provided one does not accept all the implications of the Aeby theory.

Another useful concept is the one advanced by the Russian surgeon Melnikoff (1922)—namely, that in dissecting the lung the lower lobes seem to separate into a series of oblique overlapping layers. We would restrict this to the last three basal zones (B^8, B^9, B^{10}) and add that B^7 is applied at right angles to these (Fig. 2,*B*; compare also with overlapping bronchi in Fig. 7,*B*).

1. *The Medial Basal Segmental Bronchus* (B^7).—The medial basal segmental represents the cardiac or infracardiac bronchus of mammals, which in most species occupies the space between the heart and the diaphragm. Its position on the stem is also characteristic, it being the only basal bronchus which constantly arises anteromedially (see Fig. 7). Because of this position one would anticipate that it would distribute to both the anterior and the paravertebral surfaces. Such is the case in 78 per cent of the 50 specimens examined.† Typically, it divides into an anterior (B^7a) and a medial ramus (B^7b), the line between the two falling, usually, in the sulcus of the inferior vena cava (Plate 4, No. 39).

In 14 per cent, B^7 was found to be absent as such—that is, no bronchus having that origin was distributed to that territory. In such specimens the anterior and paravertebral components of the zone were aerated by two displaced bronchi, one being an accessory branch of the anterior basal (BX^7a from B^8b) and the other an accessory branch of the subsuperior (BX^7b from B^*). In the remaining specimens (8 per cent), B^7 supplied only the anterior surface but these were peculiar in that while B^7 arose normally in one specimen (namely, Plate 3, No. 46), both of its rami were distributed to the anterior surface exclusively (see also Fig. 3,*A*). In the other three, only the anterior ramus arose in the usual position, the medial ramus originating as an accessory bronchus of the subsuperior (B^*) or of the posterior basal (B^{10}).‡

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†These observations are thus in accordance with the recent figures of Lucien and Beau (Mattei, 1946) but at variance with Jackson and Huber's account of the extent of this segment. Their figures show no distribution of the medial basal bronchus on the paravertebral surface.

‡It is interesting to note that Huntington (1920) used the cardiac bronchus of mammals as an example of the method of formation of accessory bronchi. He said, "It happens occasionally that a peripheral lung segment receives its bronchial supply from two sources. The terminal alveoli of each contributing bronchus are closed off within the segment of the district which it supplies and do not communicate with those of its colleague. Together the two components make up a topographical pulmonary district ordinarily served by a single bronchus."

TABLE VI. MEDIAL BASAL SEGMENTAL BRONCHUS (B^7)

I. B^7 present on both sides of the pulmonary ligament			78%
1. Distributes to more than one-half of the lower anterior surface		54%	
2. Distributes to one-half or less than one-half of the lower anterior surface		24%[†]	
II. B^7 wholly anterior			8%
1. Both rami present (No. 46, Plate 3; Fig. 3)		2%	
2. B^7 present but one ramus displaced		6%	
(1) Medial ramus (BX^7b) from B*	4%		
(2) Medial ramus (BX^7b) from B^{10}	2%		
III. B^7 absent as such			14%
BX^7a from B^8		14%	
BX^7b from B*	12%		
from B^{10}	2%		

[†] In one specimen (No. 43, plate 1) B^7 consisted only of a medial ramus (B^7b). BX^7a arose from B^8.

The extent of the anterior surface supplied by B^7, as measured along the diaphragmatic margin, is variable. According to Brock, "it commonly occupies less than a half of the anterior margin," but as may be seen in Table VI (Item I:1) it occupies more than one-half of the anterior surface in two-thirds of the typical specimens. If all are included that have a B^7 of typical origin (86 per cent), B^7 occupies two-thirds or more of the anterior surface in 50 per cent of specimens, one-half in 28 per cent, and only one-third or less in 8 per cent.

Its paravertebral extent is also variable, ranging from an area exceeding one-half of the vertical extent of that surface (Plate 1, No. 40) to a small zone at the extreme lower end of the lobe (Plate 2, No. 48). In only one instance did it invade the costal surface (Plate 1, No. 43). When it fails to occupy the paravertebral area or supplies only a minimal portion of it, the remainder of its territory is taken over by adjacent bronchi—B^6a (Fig. 9), or B* (Plate 2, No. 48), or a medial branch of B^{10} (Plate 3, No. 46), or any combination of these.

Of special interest are the cases in which B^7 is absent (Item III, Table VI), or wholly anterior (Item II), with the medial ramus (B^7b) developing as an accessory branch of the subsuperior. In such instances the subsuperior arises from a posteromedial position (see B*, Fig. 3, *A*), at the same level on the tree as B^7. In this position it could well be mistaken for the latter, and apparently that is the interpretation given by Nelson in Fig. 2 of his 1934 article. This led him to minimize the importance of the medial basal segment: "On the right side there is a separate cardiac branch arising from the medial wall of the lower lobe bronchus; it runs downward and slightly inwards and backwards, to supply a small area just below the root of the lung. On account of its small size, this bronchus will not be considered again."

Dévé (1900) noted an incisure on the diaphragmatic surface in 35 per cent of 180 lungs. From its position he judged that this incisure demarcated a zone corresponding to the cardiac lobe of mammals. A similar incisure of varying depth was observed in 38 per cent of the 50 specimens injected in this laboratory. In all but 2 per cent, the incisure marked the lateral boundary of the medial basal segment. Two-thirds of these were pronounced and some would have been demonstrable roentgenologically. All of them would have been noted upon surgical exploration.

In this connection, a word must be said about the relation of the pulmonary ligament to the medial basal segment. Both Dévé's Fig. 6 and Brock's Fig. 94 portray the pulmonary ligament as reflected from the lung at the posterior boundary of B^7 (see ‡, Fig. 2, *B*). In the large majority of specimens, however, the pulmonary ligament is reflected from the lung just upon or just posterior to what these authors call the "horn-like projection of the cardiac segment." In other words the right "retro-pericardiac recess" is shallow, necessitating severance of the pulmonary ligament in an interlobar approach for resection of the medial basal segment.

Because of the fissures just noted, and the superficial position of the segment, and the accessible (high) origin of the bronchus, it is felt that this zone constitutes a surgical unit which may be readily resected. Indeed, there is at least one published instance (Scannell) in which this has been successfully accomplished, for removal of a bronchiectatic segment.

2. *The Anterior Basal Segmental Bronchus (B^8).*—The anterior basal segmental bronchus is the second of the basal bronchi and arises a little below B^7 from the anterolateral aspect of the basal trunk (Figs. 3, *B* and 7). It is remarkably constant in its distribution, dividing into lateral (B^8a) and basal (B^8b) rami.

The former supplies the lower middle-anterior and lateral costal surfaces of the lobe (Plate 3, No. 46). In only 4 per cent of the fifty specimens could it be traced to the diaphragm. The upper limit of its distribution coincides with the *interfissural crest*, a more or less inconspicuous ridge on the lower lobe marking the junction of the upper and middle lobes. In 4 per cent of specimens it gives off an accessory branch (BX^6c) which extends above the interfissural crest to invade the territory of the superior segment (Plate 2, No. 42; Plate 4, No. 39).

The basal ramus (B^8b) distributes to the lower anterior (interlobar) and lower lateral costal surfaces of the lobe in addition to the anterolateral diaphragmatic surface (Plate 2, No. 48). Its extent on the interlobar surface is variable. Usually, it occupies only the lateral third (50 per cent of specimens), as measured along the inferior margin. In 16 per cent it extends medially into the sulcus of the inferior vena cava by virtue of an accessory ramus, BX^7a (Plate 1, No. 43). In one instance B^8b was displaced, occurring as an accessory ramus of B^9 (Plate 2, No. 42). In still another specimen the anterior segment occurred as a bifid lobe (Plate 4, No. 49). This was the only example of an incisure between B^8 and B^9.

In three of the four specimens in which B^9 was absent, B^8b spreads more posteriorly; in the fourth it has an accessory bronchus (BX^9b; Plate 1, No. 40).

In concluding this section, we believe that the anterior basal segment, like the medial basal, is readily resectable; for its bronchus has a high anterior origin, its distribution is fairly constant, and its medial border is frequently marked by an incisure (36 per cent of specimens).

3. *The Lateral Basal Segmental Bronchus (B^9).*—The lateral basal segmental bronchus usually arises from the anterolateral aspect of the short common stem of B^9 and ^{10} (Fig. 7, *B*). In only 10 per cent of specimens was this a long com-

mon stem. In another 8 per cent, B^{9} was entirely absent as such. We agree with Brock that it is smaller than the anterior basal (B^{8}) and appears much more like a secondary branch of the posterior basal (B^{10}) than an important independent branch. It is distributed to the posterolateral sector of the basal half of the lower lobe (Plate 2, No. 48).

Almost immediately after its origin the bronchus divides into lateral ($B^{9}a$) and basal ($B^{9}b$) rami. The lateral branch, when present (86 per cent), is quite constant both in origin and in distribution, supplying in 74 per cent of specimens the posterolateral area adjacent either to the subsuperior bronchus (Plate 4, No. 49) or to the displaced subsuperior bronchus (Plate 3, No. 46). In a fifth of this 74 per cent, the lateral ramus even encroaches upon the subsuperior zone (see Plate 3) and might be considered to contribute to that interpolated segment. An example of its infrequent lower position may be seen in Plate 2 (No. 48) and Plate 3 (No. 50). The latter is the only case in which it extended to the diaphragm. In the 14 per cent of specimens in which it is absent its place is taken by a subsuperior (10 per cent; Plate 1, No. 40) or by the lateral ramus ($B^{8}a$) of the anterior basal (4 per cent).

The basal branch ($B^{9}b$) supplies the inferior posterolateral costal area and the corresponding sector of the diaphragmatic surface (Plate 4, No. 39). In the 8 per cent of specimens in which it was absent its place was taken by $B^{8}b$—by spread of that ramus in three instances, and by an accessory branch of that ramus ($BX^{9}b$) in one instance (Plate 1, No. 40).

4. *The Posterior Basal Segmental Bronchus* (B^{10}).—The posterior basal segmental bronchus appears to be the continuation of the right lower lobe bronchus, particularly the branch here designated as $B^{10}b$ (Figs. 3 and 7) which extends to the inferior posteromedial corner of the lobe. In dissecting specimens it has been found most useful to locate this ramus first, then the other branches may be quickly identified.

Typically B^{10} breaks up into three major branches. The highest (formerly numbered $B^{10}a$, Boyden 1945) has already been demonstrated to be in reality a displaced subsuperior, and accordingly has been redesignated $BX^{*}(10)$. It will be recalled that this is a fairly constant branch occurring in 86 per cent of specimens (see Table III).

Having disposed of this high dorsal branch (from Aeby's level 3), let us now consider the two major branches of the posterior basal, herewith designated the laterobasal ($B^{10}a$) and mediobasal ramus ($B^{10}b$).† In over one-half the specimens (58 per cent), $B^{10}b$ arises somewhat behind $B^{10}a$, but in virtually all cases both rami are distributed to both the costal and diaphragmatic surfaces. In addition, $B^{10}b$ is distributed to the paravertebral surface (for example, Plate 4, No. 49). Occasionally (10 per cent), a particular paravertebral branch of B^{10} may be noted, but it is not constant enough to merit a name.

SUMMARY

The distribution and arrangement of the segmental bronchi of the right lower lobe have been studied in fifty fresh lungs by the following methods: (1) the successive injection of the principal bronchi by differently colored gelatin masses; (2) the accurate sketching of each injected lobe, to record the surface distribution of the bronchi on interlobar, costal, and diaphragmatic surfaces; (3) the dissection of each lobe, to verify the distribution of bronchi, and to ascertain their mode of branching.

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†These are the rami formerly designated by Boyden (1945) as $B^{10}b$ and $B^{10}c$.

From these data it has been possible to establish the prevailing pattern empirically, and to analyze the principal variations in the light of that knowledge.

As viewed from the interlobar surface the right lower lobe may be divided into superior and basal zones, the line of separation usually falling at the interfissural crest. Posteriorly, an interpolated, "subsuperior" zone can be demonstrated between superior and basal segments in 100 per cent of specimens (vide infra).

The Superior Segment.—Since the superior segment is resectable, it is important to know its extent. Prevailingly, it "caps" the basal segments horizontally, but in 38 per cent of specimens it is obliquely fitted to the basal segments. Usually (in 78 per cent), it occupies the upper half of the lobe, but it may occupy two-thirds of the lobe (8 per cent) or only one-third (14 per cent). On the paravertebral surface it may extend two-thirds (or more) of the way to the diaphragm (30 per cent of specimens).

The superior segmental bronchus (B^6) has three constant rami—a medial (B^6a), a superior (B^6b), and a lateral ramus (B^6c)—but it rarely trifurcates (6 per cent), the prevailing pattern (86 per cent) being a bifurcation into $B^6a + b$ and B^6c. On the interlobar surface the superior segment is usually fused to a greater or lesser degree with the right upper lobe; and in five out of eleven consecutive specimens examined for this purpose a branch of B^6b was observed to supply the adjacent portion of the right upper lobe. This may explain how consolidation of the lower lobe, in lobar pneumonia, may extend into the adjoining area of the upper lobe.

The *subsuperior* zone is a constant posterior zone interpolated between superior and basal segments. In this series of dissections it has been identified in 100 per cent of specimens, as contrasted with Brock's identification of only 48 per cent in a series of bronchograms. It is supplied by one or more posterior bronchi of inconstant origin. These arise from levels 1.5 to 5 cm. below the orifice of the superior bronchus (B^6). For this reason, the bronchi supplying this zone have been indicated by the noncommittal designation, "B^*" (Boyden, 1945).

In general the principal levels of origin correspond to Aeby's dorsal bronchi, d_2 and d_3 (the first dorsal being the superior bronchus, B^6). That posterior bronchus which arises from Level 2 (that is, from the common stem of $B^{8,\ 9,\ \text{and}\ 10}$ or B^{9+10}) is designated the "subsuperior proper," B^*. That posterior bronchus which arises from Level 3 (that is, from the trunk of B^{10}) is designated the "accessory subsuperior," $BX^*(10)$.

In about one-half the specimens (48 per cent), the subsuperior bronchi arise from both levels, and range in number from two to four. In the remaining one-half (52 per cent), there is only one subsuperior bronchus—more commonly BX^* (38 per cent), as against 14 per cent with a subsuperior proper.

Primarily the subsuperior zone lies in the posterior sector of the lobe, but it may spread laterally or medially. It is a common site of lung abscess.

The *basal bronchi* are four in number and tend to radiate from above downward into the anteromedial, anterolateral, posterolateral, and posteromedial sectors of the lobe, respectively.

The *medial-basal segmental bronchus* (B^7) is the highest in origin. It distributes to the zone that lies on either side of the pulmonary ligament (78 per cent of specimens), and is placed athwart the other basal zones—that is, it occupies a plane virtually at right angles to the others. This segment corresponds to the cardiac lobe of mammals. Therefore, it is not surprising to find an incisure of varying depth separating it from the anterior-basal segment in 36 per cent of specimens. In two-thirds of this number the incisure is possibly deep enough to have been visible in a roentgenogram.

The bronchus to this "cardiac" region commonly divides into an anterior (B^7a) and a medial ramus (B^7b). The line between the two usually falls in the sulcus of the inferior vena cava.

In 14 per cent of specimens the medial basal bronchus is absent as such, the zone being supplied by one or two accessory rami from the adjacent bronchi—B^8, B^*, or B^{10}.

Contrary to Brock, the zone prevailingly occupies two-thirds or more of the anterior margin of the lobe; and contrary to Jackson and Huber it usually extends onto the paravertebral surface.

Because of the high accessible origin of its bronchus, the frequent presence of incisures and the superficial position of this segment, it is deemed suitable for resection.

The *anterior basal segmental bronchus* (B^8) is remarkably constant. It arises from the anterolateral aspect of the basal trunk, a little below B^7. It soon divides into a lateral (B^8a) and a basal ramus (B^8b). The former supplies the area on either side of the interlobar margin below the level of the interfissural crest. The latter supplies the lower part of this sector including the corresponding diaphragmatic area. Usually (50 per cent), B^8b occupies only the lateral one-third of the lower interlobar surface as measured along the inferior margin of the lobe, but in 16 per cent of specimens it extends as far medially as the sulcus of the inferior vena cava. In another 8 per cent it takes over the territory of B^9b. Occasionally (4 per cent), the lateral ramus (B^8a) invades the superior segment.

Because of the high origin of B^8, its fairly constant distribution, and the fact that its medial border is frequently marked by an incisure (36 per cent) it, too, is deemed suitable for resection.

The *lateral basal segmental bronchus* (B^9) arises deep within the lobe and is not considered to be readily resectable. It is absent in 8 per cent of specimens. Typically it divides almost immediately after its origin into a lateral (B^9a) and a basal bronchus (B^9b). The former ramus, when present (86 per cent), supplies the posterior lateral zone adjacent to the subsuperior. When absent, its territory is supplied by B^* or B^8a. The basal ramus (B^9b) supplies the lower posterior lateral sector and the corresponding diaphragmatic area. When absent (8 per cent), its territory is taken over by B^8b.

The *posterior basal segmental bronchus* (B^{10}) appears to be a continuation of the lower lobe bronchus—particularly the ramus $B^{10}b$ which extends into the inferior, posteromedial corner of the lobe. Typically, B^{10} breaks up into three major branches. The highest is that which has been already described as the accessory subsuperior, $BX^*(10)$. It occurs in 86 per cent of specimens and is reflected upward or backward to supply the subsuperior segment. The two remaining major branches (the mediobasal, $B^{10}b$, and the laterobasal ramus $B^{10}a$) pass respectively to the paravertebral and posterior surfaces, and to the corresponding diaphragmatic sectors.

Because of the inaccessibility of its origin, this bronchus, like B^9, is not considered to be readily resectable.

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AN ANALYSIS OF VARIATIONS OF THE SEGMENTAL BRONCHI OF THE LEFT LOWER LOBE OF FIFTY DISSECTED, AND TEN INJECTED, LUNGS

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INTRODUCTION

A SURVEY of the literature of the bronchopulmonary segments leaves one with the impression that the left lower lobe of the lung is a somewhat condensed or even defective version of the right lower lobe: that if one understands the pattern of the right side it is necessary to remember only one or two modifications—such as the union of medial and anterior basal bronchi—to comprehend the left.

Such at first was our experience. Soon, however, it began to appear that the left lobe, although capable of being compared with the right, is distinctive throughout. Thus, for instance, the rami of the left superior segment have a different system of branching, the left subsuperior zone is prevailingly posterolateral and not posterior, the left anterior basal and not the left medial basal is the "defective" bronchus, the lateral branch of the left lateral basal contributes to the subsuperior zone, and the left posterior basal, in a third of the specimens, is characterized by a prominent paravertebral ramus.

Hitherto, only Brock (1942 to 1944) seems to have made any detailed study of the variations of the lower lobe segments and he has largely confined himself to describing the varying extent of the segmental bronchi. Our purpose has been to establish the prevailing pattern in a large number of specimens and from this firm and hitherto unestablished base to analyze the significant and puzzling variations which are so frequently encountered.

In general, the terminology of Jackson and Huber (1943) has been employed, supplemented by the system of enumeration of bronchi developed by Boyden (1945). Also, throughout the article, the segmental pattern of the left lower lobe has been compared with that of the right, as recently presented by Smith and Boyden (1949).

MATERIAL AND METHODS

This study is based upon two sets of data: (1) a detailed dissection of fifty preserved specimens (R.M.B.) and (2) colored gelatin injections of ten fresh specimens (F.R.S.).

In the first group, each lobe was dissected from the anterior surface and then accurately drawn. Supplementary sketches recorded the projection of basal

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bronchi on the diaphragmatic surface, the bronchoscopic appearance of the orifices (here inverted), and the sectors supplied by various bronchi—particularly the subsuperiors (for example, text Figs. 5 and 6).

In the second group, fresh specimens were injected with colored gelatins (after the method employed in the study of the right lobe, Smith and Boyden, 1949), and then allowed to harden only one day or so before being dissected and drawn for publication (E.A.B.), for example, Plates 1 to 4.

By means of these two methods—dissection, and injection followed by dissection—it has been possible to establish the prevailing patterns and to display the common variations in the branching and surface distribution of the segmental bronchi.

THE GROSS CONFIGURATION OF THE LEFT LOWER LOBE

The left lower lobe has the shape of a cone, the upper part of which has been compressed. It is divided for descriptive purposes into four surfaces—namely, anterior, costal, paravertebral, and diaphragmatic.

The first two are separated by the sharp interlobar margin (Fig, 4, *B*, and *X*, Fig. 3), the second and third by an ill-defined obtuse margin—the costoparavertebral—which occupies the interval between the heads and tubercles of the ribs (Fig. 4, *B* and *Y*, Fig. 3), and the anterior and paravertebral by the pulmonary ligament and hilum.

The *anterior surface* (middle column of figures, Plate 1) is not a flat plane but exhibits a shallow S-shaped curve—that is, it is molded in such a way that the upper portion faces somewhat laterally and the lower portion somewhat medially. Usually, the lower portion is subdivided into cardiac and interlobar areas by the frenum* of the upper lobe (see Plate 1, No. 52). Usually, as in this plate, the frenum leaves the margin of the hilum between the superior and inferior pulmonary veins, but it may originate anywhere along the pulmonary ligament.† In several of the injected specimens it was coextensive with the pulmonary ligament, and the upper lobe was fused to the cardiac area of the lower lobe. In such cases (Plate 4) this lingular portion of the upper lobe was aerated by small branches of the medial basal bronchus.

The *costal surface* presents a smoothly rounded contour which may be divided arbitrarily—for the purposes of describing segments—into posterior, posterolateral, and lateral sectors (Fig. 4, *B*).

The *paravertebral surface* (Fig. 4, *B*) is molded to the posterior mediastinum and is markedly grooved by the descending aorta. As noted in the foregoing, the costoparavertebral margin is obtuse. For the purpose of describing segments, its position in the plates is indicated by a solid line (Plate 1, left row of figures).

In the course of this study various degrees of fusion between upper and lower lobes were observed. In the literature there is frequent mention of this condition—for example, Kent and Blades (1942) stated that the interlobar

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*A term introduced by Churchill and Belsey (1939).

†For more extended discussion, see article by Boyden, E. A.: Cleft Left Upper Lobes and the Split Anterior Bronchus, Surgery, 1949. (In press.)

EXPLANATION OF PLATES

(Sketches rendered for publication by Lawrence B. Benson)

Each plate contains five drawings each, of two injected specimens of the left lower lobe. The large figures at the left depict the costal surface of the lower lobe, the large central ones the anterior surface, and the large figures on the right the dissected anterior surface (each X ¼). In addition, there is a reduced drawing of the diaphragmatic surface (X ⅛) and a small sketch of the orifice of the basal trunk.

Yellow color indicates distribution of superior segmental bronchus (B^6); *white*, the subsuperior zone (B^* and BX^*); *blue* (primarily on anterior surface), the distribution of the medial basal segmental bronchus (B^7); *red*, the distribution of the anterior basal segmental bronchus; *blue* (primarily on costal surface), the distribution of the lateral basal segmental bronchus (B^9); *green*, the distribution of the posterior basal segmental bronchus (B^{10}). *Stippled* areas indicate cut surface of left upper lobe where it is fused with left lower lobe.

Plate 1.—*Specimen 52*, This is characteristically capped by an obliquely placed superior segment (B^6). Typical, also, is the subsuperior zone (B^*), in that it occupies the posterolateral sector of the costal surface. Its extension into the posterior sector is not typical (see Table IV). The basal segments demonstrate the prevailing pattern except that the anterior basal segmental bronchus (B^8) fails to reach the diaphragmatic surface. The two dorsal rami of the posterior basal, labeled (BX^*?), represent accessory subsuperiors which would have been branches of the *main* trunk of B^{10} had the latter not bifurcated so high (see $BX^*[10]$ in No. 51).

Specimen 51, This is atypical in that B^6a2, the deep branch of the medial ramus of the superior bronchus, has displaced the subsuperior bronchus (B^*) lateralward, on the costal surface. Also the stem of B^{7+8} seems to have rotated medially, leaving that portion of the anterior surface which is usually supplied by B^8a to be taken over by accessory rami (BX^8a) of the subsuperior (B^*) and of the lateral basal (B^9). Accordingly, B^8 (and, to a lesser extent, B^7) supplies a much smaller than usual area.

Plate 2.—*Specimen 56*, This illustrates a subsuperior zone which is supplied by accessory subsuperior bronchi $BX^*(10)$ and $BX^*(9)$, from both the posterior and the lateral basal bronchi respectively. Since these usually arise at the top of their respective bronchi, the tip of the cannula usually projects beyond their orifices leaving the zone uninjected, as in this specimen. This lobe is of further interest in that the superior segment is partially separated from the basal segments by a fissure.

Specimen 58, This very much resembles No. 51 (Plate 1), in that B^6a2 has displaced the subsuperior zone laterally. (In this specimen the zone is supplied by an accessory subsuperior, BX^*, from the *posterior* basal, B^{10}.) Incidentally, there are two accessory subsuperiors from B^{10}, the second not quite reaching the subsuperior zone. Frequently, as here, a $BX^*(10)$ may arise conjointly with a paravertebral ramus of B^{10}. Also the anterior basal (B^8) encroaches on the medial basal (B^7), but in this case B^7a is present.

Plate 3.—*Specimen 55*, This has a subsuperior zone which is almost a duplicate of that in No. 53 except that it is supplied by two accessory subsuperiors from different sources—namely, high branches of both B^{10} and B^9. The anterior basal (B^8) is represented by both of its rami, but again fails to reach the diaphragmatic surface. Here the medial basal is separated from the other basal segments by a deep fissure, best shown on the diaphragmatic surface. Such fissures are not uncommon (25 per cent).

Specimen 53, Here the subsuperior zone is supplied by a high accessory branch (BX^*) of B^{10} which is almost high enough to be a subsuperior proper. Also, it distributes to the posterior as well as the posterolateral sector of the zone, since B^6a2 is absent. Also in this specimen, the basal ramus of the anterior basal (BX^8b) arises as an accessory branch of the lateral basal (B^9). Therefore B^8 appears to be restricted to a small area on the midanterior surface (B^8a).

Plate 4.—These specimens exhibit a fusion of the upper lobe with the lower lobe, the area of fusion extending a varying distance laterally from the whole length of the pulmonary ligament. In each case the thin portion of the lower lingular segment (B^5) was aerated by small accessory branches (BX^5) of the medial basal (B^7). The specimens are also similar in that the basal ramus of B^8 arises as an accessory branch (BX^8b) of the medial basal (B^7).

Specimen 60, Here the subsuperior zone is very extensive, being supplied by a large subsuperior proper (B^*) and two accessory subsuperiors $BX^*(9)$ and $BX^*(10)$. The last of these arises in conjunction with a paravertebral branch of the posterior basal. Also the lateral ramus of the superior segment (B^6c) has what appears to be displaced branches of the superior ramus (B^6b?). This is the "cluster type" of branching noted in Table I.

Specimen 54, This is characterized by three accessory subsuperiors (BX^*), one from the top of the lateral basal (B^9), and two from different levels on the posterior basal (B^{10}). Although one of the last two arises some distance below the other, they both distribute to the same level. The zone as a whole is somewhat depressed by a large B^6a2. In this specimen, the medial ramus of the medial basal arises as an accessory branch of B^9 (see BX^7b).

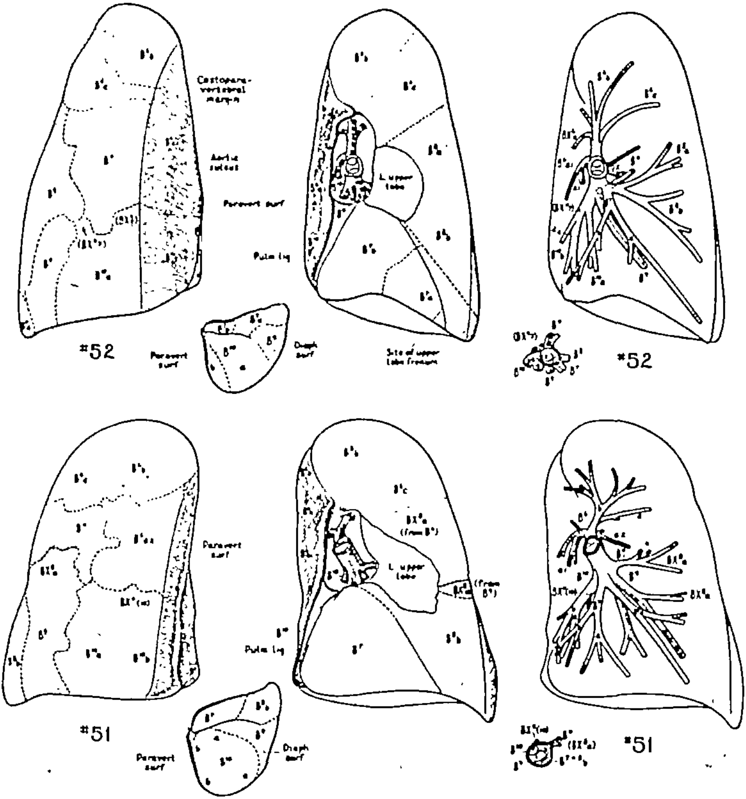

Costoparavertebral margin
Aortic sulcus
Paravert surf
Pulm lig
L upper lobe
Site of upper lobe fissure
Paravert surf
Diaph surf
#52
Paravert surf
L upper lobe
Pulm lig
Paravert surf
Diaph surf
#51

Plate 1.

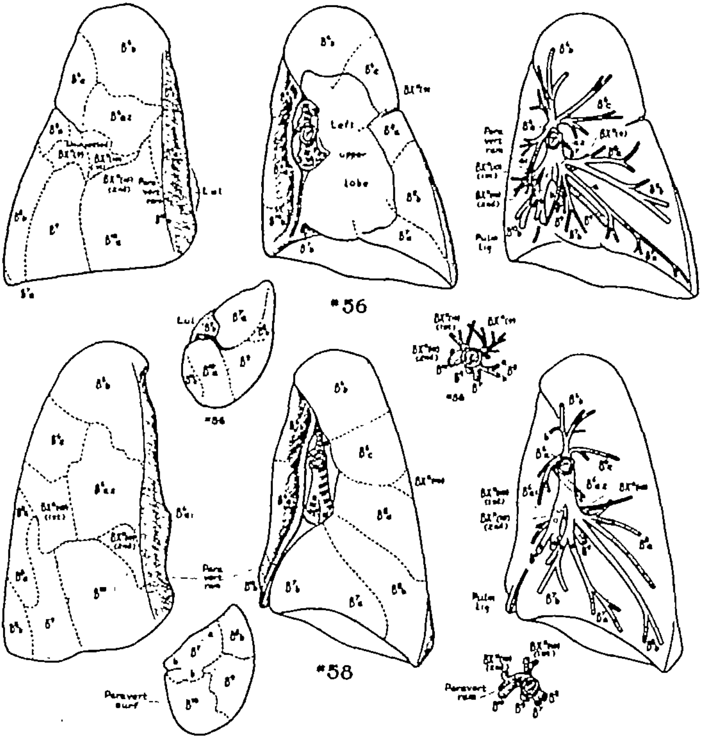
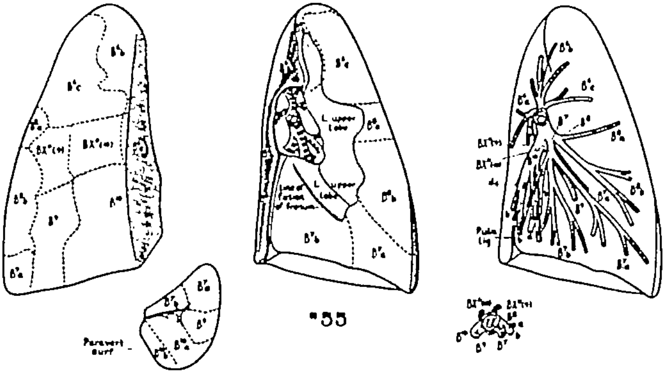
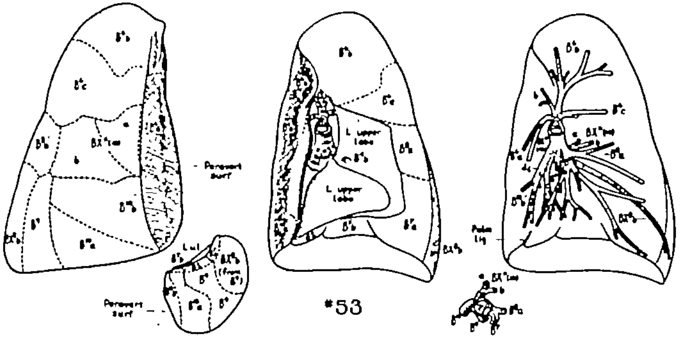

#56
#58
Left upper lobe
Lat
Para vert ram
Pulm lig
Paravert surf
Paravert ram
Unaerated

Plate 2.


L upper lobe
L upper lobe
Line of fusion of fissure
Pulm lig
Paravert surf

*55


Paravert surf
L upper lobe
L upper lobe
Pulm lig
Paravert surf

*53

Plate 3.

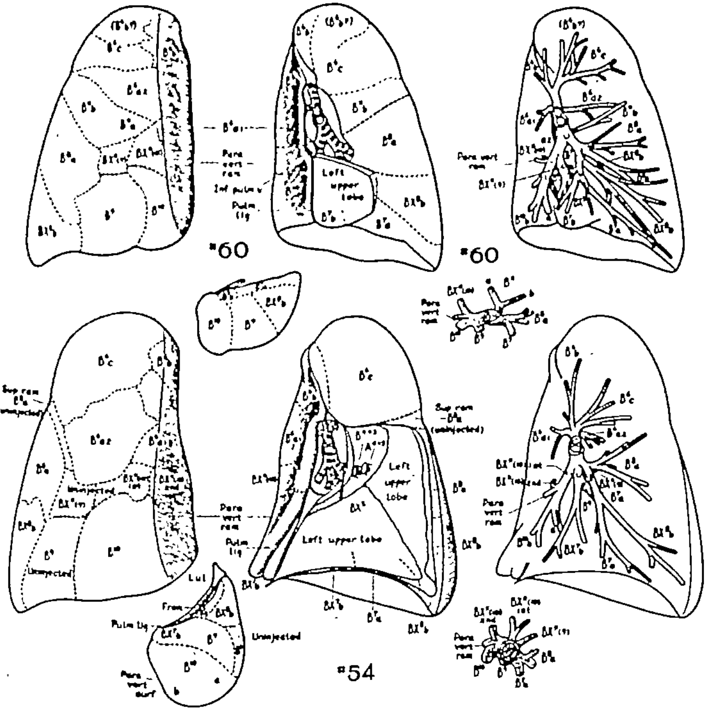

#60
#60
#54
Para vert ram
Inf pulm v
Pulm lig
Left upper lobe
Sup ram
(uninjected)
Unlnjected
Lat
Pulm lig
Para vert surf
Left upper lobe

Plate 4.

fissure was incomplete in about 30 per cent of 277 lungs, being equally common on each side.

THE SUPERIOR SEGMENTAL BRONCHUS (B^6)

The superior bronchus arises just below the upper lobe bronchus in such a position as to complicate resection of the lower lobe, that is, its keel lies only 1 cm. below the keel of the lower lobe bronchus. It is the highest dorsal branch of the lower lobe and therefore has been considered by Brock to be the principal site of lung abscess in this lobe. As on the right side, it breaks up into three constant rami—the medial branch (B^6a) running medially and downward, the superior branch (B^6b) running upward, and the lateral branch (B^6c) coursing laterally and somewhat downward.

In mode of branching the prevailing pattern of B^6 agrees with that of the right side in being bifurcate, but differs in the formula. On the left it usually divides into B^6a and $B^6b + c$ (43 per cent of specimens; Fig. 1, No. 37); on the right it divides into B^6c and $B^6a + b$ (86 per cent).* The percentages indicate that the branching on the left side is more variable than on the right, while the lower and independent origin of B^6a suggests that the paravertebral extent of the bronchus will be much greater on the left side (vide infra).

The frequency of the various types of branching is indicated in Table I. The type recorded in Item I: 2 (see Fig. 1, No. 4) is the one considered most "normal" by Brock (1943). That in Item I: 3 (see Fig. 1, No. 48) is the one described for the left side by Ewart (1889). This is the prevailing pattern on the right side. No one seems to have observed what is termed the "cluster type" (Item I: 4, see Plate 4, No. 60). Here, B^6b is represented by more than one main branch.

Trifurcation of B^6 is relatively uncommon (15 per cent; Fig. 1, No. 55), but not so rare as on the right side (6 per cent).

TABLE I. THE SUPERIOR SEGMENTAL BRONCHUS (B^6)

I. Bifurcates Into:			85%
1. B^6a and $B^6b + c$	(Fig. 1, No. 37)	43%	
2. B^6b and $B^6a + c$	(Fig. 1, No. 4)	27%	
3. B^6c and $B^6a + b$	(Fig. 1, No. 48)	10%	
4. Cluster type		5%	
(1) $B^6a + b$ and $B^6b + c$			
(2) B^6a and $B^6b + (B^6b + c)$	(Plate 4, No. 60)		
(3) B^6b and $(B^6b + c) + B^6a$			
II. Trifurcates Into B^6a, B^6b, B^6c	(Fig. 1 and Plate 3, No. 55)		15%

The distribution of the superior bronchus is much like that on the right side but differs in at least two particulars. First, in the majority of left lower lobes the superior segment caps the basal segments very obliquely, thus presenting an anatomic hazard to the resection of this segment. (On the right the segment is horizontally placed in 62 per cent.) This is due to the fact that the medial ramus (B^6a) extends downward along the paravertebral surface to within a short distance of the diaphragmatic surface—specifically, it reaches the lower third or less of the paravertebral surface in 57 per cent of specimens (for example, Plate 3, No. 53).*

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*The statistics for the right side which will be referred to frequently are those published by Smith and Boyden (1949).

Second, in 45 per cent of left lower lobes, there is a large posterior branch (B^6a2) which takes over the posterior sector of the subsuperior zone. It is represented occasionally on the right side by a deep branch of B^6a, but on the left it is so striking as to merit a special designation (B^6a2, Fig. 1, No. 37 and Plate 4). In certain specimens in which B^6a2 is absent, its territory is taken over by a large, posterior descending branch of B^6c as in Fig. 1, No. 48. (See discussion of subsuperior zone.)

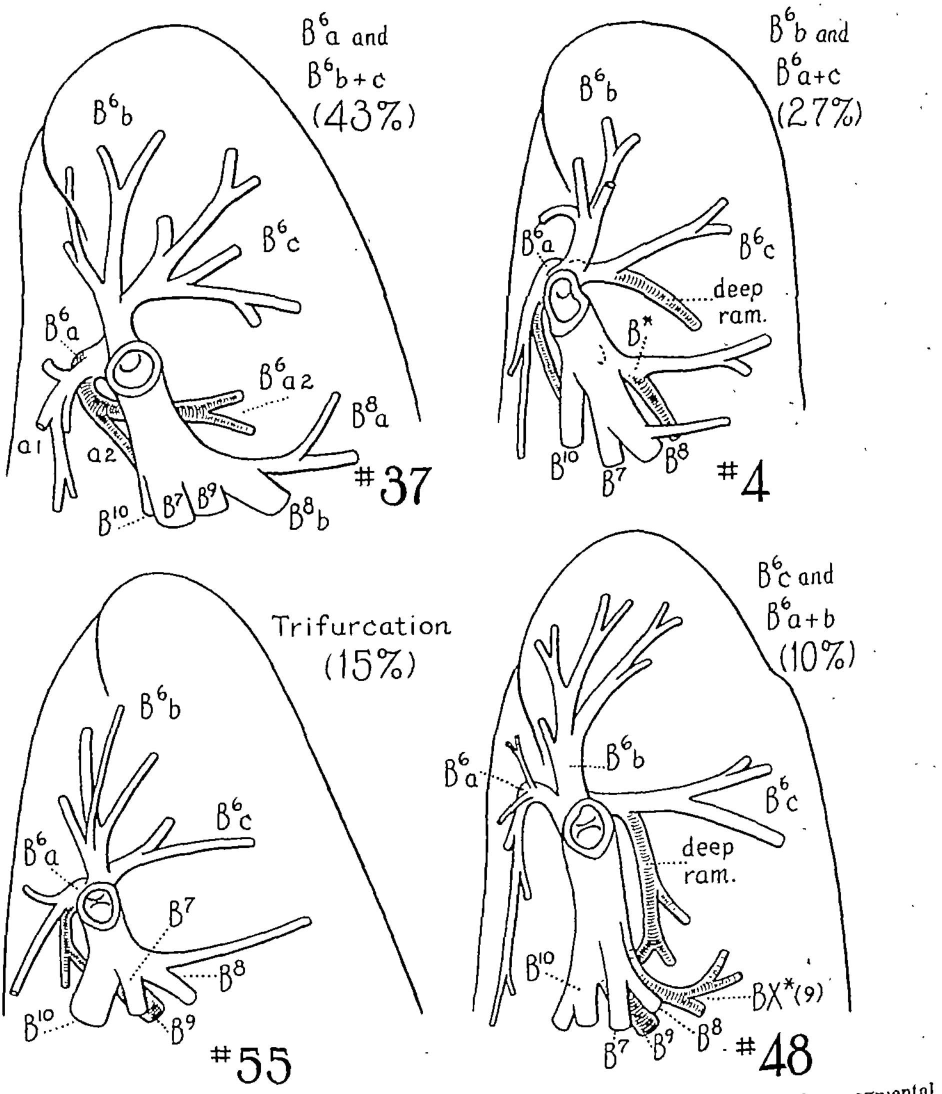

Fig. 1.—Selected drawings illustrating mode of branching of the superior segmental bronchus (X ⅓). (B^6a, medial ramus; B^6b, superior ramus; B^6c, lateral ramus.) In addition, specimen No. 37 shows a deep branch of ramus *a* (B^6a2) which occurs in 45 per cent of specimens; No. 48 illustrates a deep branch of ramus *c* (*deep ram.*), which occurs in 18 per cent of specimens.

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*This finding is, therefore, at variance with the statement of Huizinga and Behr (1940) that "the lower limit of this dorsal segment is always horizontal."

B^6a is of further interest in that in 13 per cent of specimens, part of its paravertebral territory is taken over by an accessory branch of the superior ramus (for example, BX^6a, Fig. 6, No. 23; Plate I, No. 52); in another 2 per cent the lower two-thirds of the paravertebral surface is taken over by a high accessory branch of B^{10}. The distribution of these accessory branches is indicated in the footnote to Table II.

TABLE II. LOWEST EXTENT OF MEDIAL RAMUS (B^6a) ON THE PARAVERTEBRAL SURFACE

Two-thirds or more of distance to diaphragm (Plate 3, No. 53)	57%*
Halfway to diaphragm (Plate 4, No. 60)	37%
One-third of distance to diaphragm	3%
Fails to reach paravertebral surface and is replaced by a branch of B^6b	3%

*In 8 per cent the upper portion of paravertebral surface is replaced by an accessory branch of B^6b. Since this occurs altogether in only 15 per cent of specimens it would seem that when the medial ramus descends the lowest there is a tendency for its upper territory to be taken over by B^6b.

Discussion of this segment should not be concluded without mentioning the occurrence of a notch on the lateral side of the interlobar margin of the lobe which marks the separation of the superior from the basal segments in 5 per cent of specimens (Fig. 6, No. 8; Plate 2, No. 56) and of an extensive fissure recently observed in an autopsy specimen. This is a much lower frequency than was reported by Dévé in his study of 180 infants (14 per cent on the left, 30 per cent on the right, 12 per cent bilateral). Pierret, Coulouma, Breton, and Devos (1938) did not give separate figures for the two sides but reported the occurrence of the fissure in 20 per cent of specimens. Nelson (1934) considered that this inconstant fissure provided supporting evidence for his classification of the superior segment as a separate lobe, but it would seem that the fissure is no longer necessary to the argument.

THE SUBSUPERIOR BRONCHUS (B^*)

On the right side the subsuperior bronchus has been identified as the second dorsal ramus of Aeby, that one which arises from the stem bronchus between the first dorsal or superior bronchus (B^6) and the origin of B^9 (B^*, Fig. 2, *A*). On that side, it usually supplies the region which occupies the interval between the superior and the posterior basal segments in the posterior sector of the costal surface. On the left side, we will show that it arises from the same portion of the stem bronchus (B^*, Fig. 2, *B*), is distributed to the same horizontal level, but that it supplies the posterolateral sector.

Neil, Gilmour and Gwynne (1937) were the first modern investigators to call attention to this "site of localized pathological change—an anatomical gap in the distribution between the apical [B^6] and paravertebral [B^{10}] bronchi." When this bronchus was absent they noted that the "subapical segment" was supplied by a branch of the posterior basal (B^{10}). Smith and Boyden (1949) confirmed these observations and named the two types of bronchi that supply this intermediate zone the *subsuperior proper*, B^*, and the *accessory subsuperior*, $BX^*(10)$.[†] Because of the prevailingly dual and variable bronchial supply,

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[†] Interpolation of the letter "X" indicates that the bronchus supplying a given zone arises from another portion of the bronchial tree than is customary. The figure in parentheses tells its new site of origin. Thus "$BX^*(10)$" indicates that the subsuperior zone is supplied by a high branch of B^{10} instead of by the second dorsal branch of the stem bronchus.

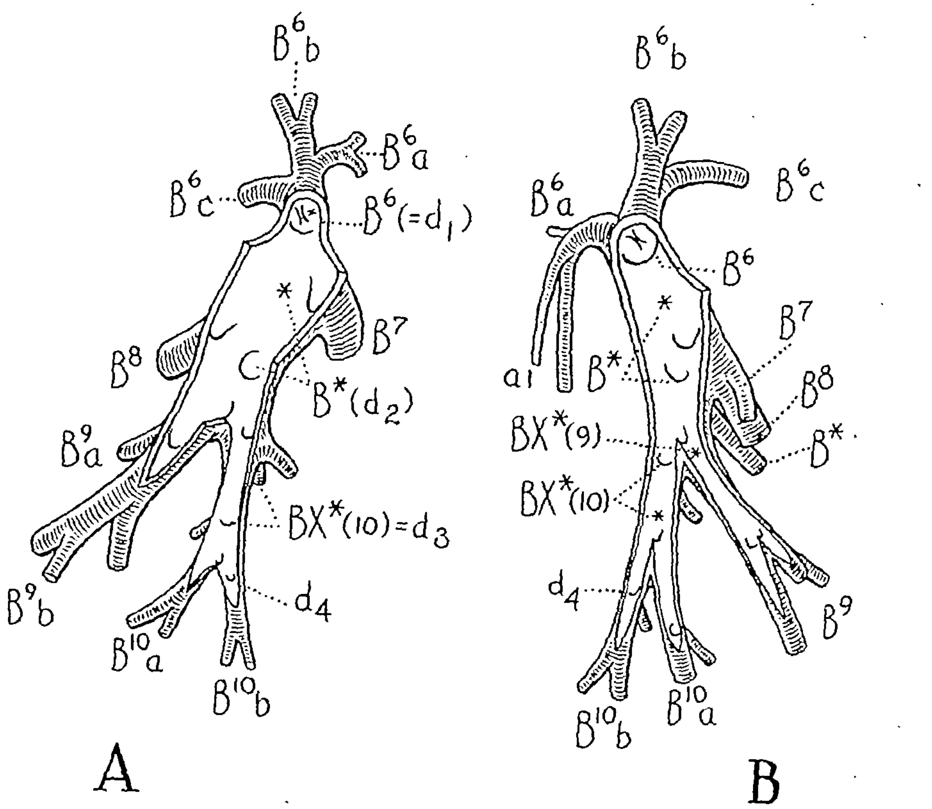


Fig. 2.—Sketches comparing the sites of origin of dorsal branches of the stem bronchus in left and right lower lobes. *B*, drawing of left side (specimen No. 60). *A*, drawing of right side after Smith and Boyden (1949). The specimen in *A* has five dorsal rami; the site of a possible sixth is noted by an asterisk. These have been grouped into four levels, corresponding to the four dorsal bronchi postulated by Aeby, in 1880. The first level (d_1) is the site of the superior segmental bronchus (B^6); the second (d_2), the site of the subsuperior proper (B^*); the third (d_3), the site of the accessory subsuperior (BX^*). The orifices of corresponding dorsal bronchi on the left side are shown in B. The principal difference between the two sides as that on the left the dorsolateral branch of B^9 (BX^*[9]) also participates in the subsuperior zone (see discussion in text).

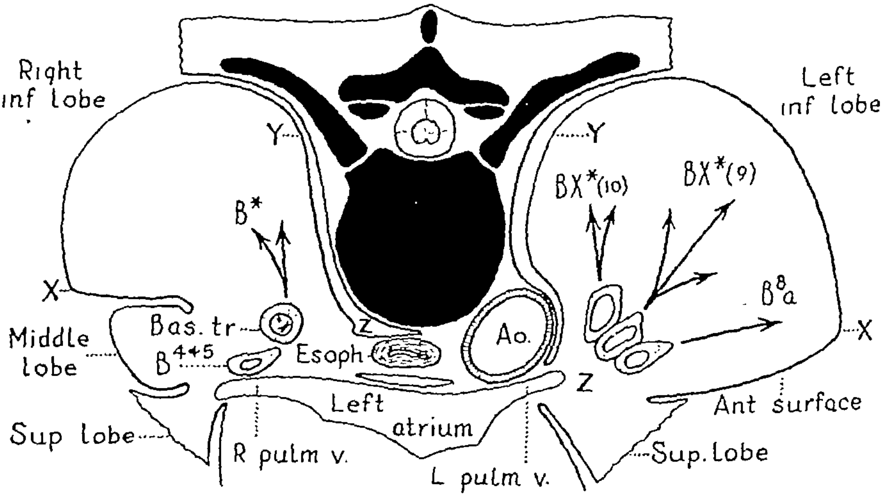


Fig. 3.—Transverse section of the lower lobes of an intact thorax to illustrate the differing direction of the subsuperior bronchi on the two sides. On the right, the subsuperior proper (B^*) distributes primarily to the posterior sector of the costal *surface; on* the left, it may distribute to the posterior sector (see BX^* from B^{10}), but prevailingly it has a posterolateral distribution (see BX^* from B^9). $BX^*(10)$ and $BX^*(9)$ are *left accessory subsuperiors, arising* just beneath the left subsuperior proper (B^*), which does not appear in this figure. *X*, interlobar margin of lower lobe; *Y*, costoparavertebral margin; *Z*, hilum (or, lower down, the pulmonary ligament).

they did not follow Neil in giving this zone the status of a segment, or numbering it—hence the noncommittal designation B^{*}.

On the left side, this intermediate zone is equally conspicuous and clinically significant, but the bronchi supplying it are more numerous. In a later article (1939) Neil and his co-workers stated that the subapical bronchus [B^{*}] "may be absent on the left side, in which case we have found a branch going to its customary distribution arising from the axillary basal [B^{9}] or from the posterior basal [B^{10}]." With this conclusion we would also agree, but it would seem that the evidence supporting it has yet to be presented.

In the first place, analysis of the 60 specimens indicates that on the left side, as contrasted with the right, the subsuperior proper (B^{*}) always distributes to at least the posterolateral sector and most commonly to both the posterolateral and lateral sectors of this intermediate zone (see Table IV, Item IV), whereas on the right side it is distributed primarily to the posterior sector (B^{*}, Fig. 3). Furthermore, the angle it makes with the anterior surface is prevailingly 40 to 45 degrees (23 per cent; see Table IV, Item III: 1).

For this reason, both the uppermost dorsal branches of B^{9} and B^{10} that enter into the composition of the subsuperior segment must be considered acces-

TABLE III. OCCURRENCE OF SUBSUPERIOR BRONCHI (B^{*} AND BX^{*})

				In 50 rt. lower lobes‡
I. Frequency of Occurrence in 60 Left Lower Lobes†				
1. Origin exclusively from level d_1 (as branch of main lower lobe bronchus between the superior and basal bronchi)			7%	14%
2. Origin exclusively from level d_2 (as an accessory subsuperior, $BX^{*}(10)$ or $BX^{*}(9)$)			71%	38%, $BX^{*}(10)$
3. Origin from both levels in same specimen			22%	48%
			100%	100%
II. Detailed Analysis				
1. Subsuperior represented by single bronchus			22%	50%
(1) Arising at d_1 level (as B^{*})		7%		(14%)
(2) Arising at d_2 level (as BX^{*})		15%		(34%)
(a) From top of B^{10}	8%			
(b) From top of B^{9}	7%			
2. Subsuperior represented by two bronchi			61%	34%
(1) One from level d_1(B^{*}) and one from level d_2(BX^{*})		13%		(32%)
(a) B^{*} and $BX^{*}(10)$	11%			
(b) B^{*} and $BX^{*}(9)$	2%			
(2) Two from level d_2 (BX^{*})		48%		(2%)
(a) $BX^{*}(9)$ and $BX^{*}(10)$	43%			
(b) Two $BX^{*}(10)$	5%			
3. Subsuperior represented by three bronchi			17%	14%
(1) Two from level d_1(B^{*}) and one from level d_2, $BX^{*}(10)$		2%		
(2) One from level d_1(B^{*}) and two from level d_2, $BX^{*}(10)$ and $BX^{*}(9)$		7%		
(3) Three from level d_2 Two $BX^{*}(10)$ and one $BX^{*}(9)$		8%		
4. Subsuperior represented by four bronchi (two from each level)			0%	2%
			100%	100%

†Percentages in round numbers.

‡Data from Smith and Boyden (1949).

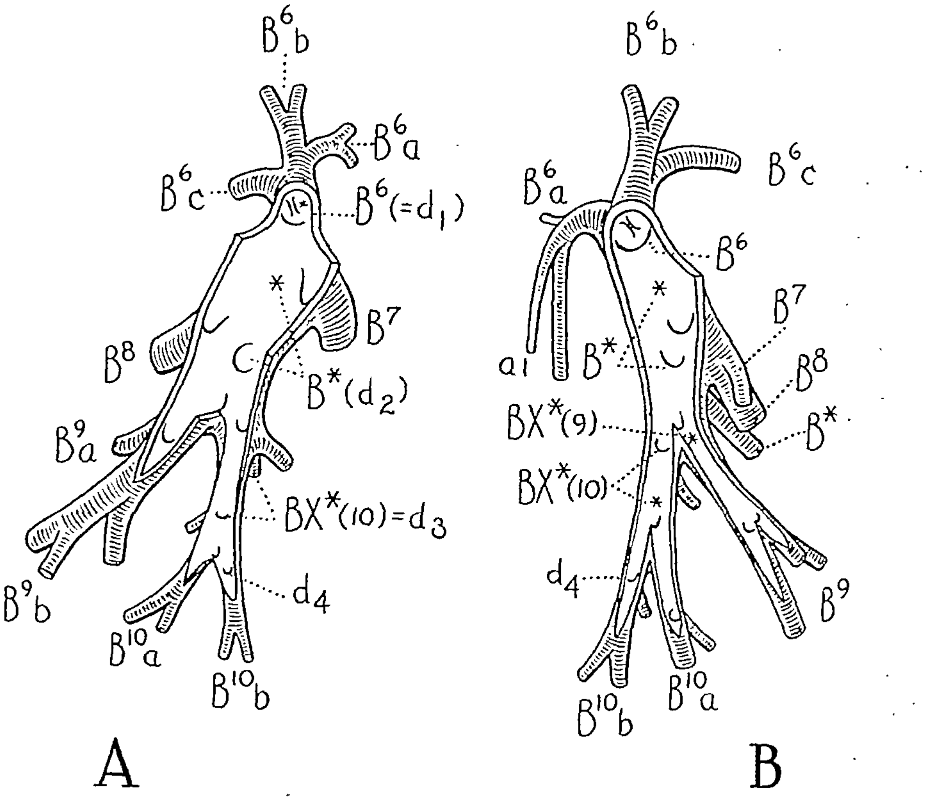


Fig. 2.—Sketches comparing the sites of origin of dorsal branches of the stem bronchus in left and right lower lobes. *B*, drawing of left side (specimen No. 50). *A*, drawing of right side after Smith and Boyden (1949). The specimen in *A* has five dorsal rami; the site of a possible sixth is noted by an asterisk. These have been grouped into four levels, corresponding to the four dorsal bronchi postulated by Aeby, in 1880. The first level (d_1) is the site of the superior segmental bronchus (B^6); the second (d_2), the site of the subsuperior proper (B^*); the third (d_3), the site of the accessory subsuperior (BX^*). *The orifices* of corresponding dorsal bronchi on the left side are shown in B. The principal *difference* between the two sides as that on the left the dorsolateral branch of B^9 ($BX^*[9]$) also participates in the subsuperior zone (see discussion in text).

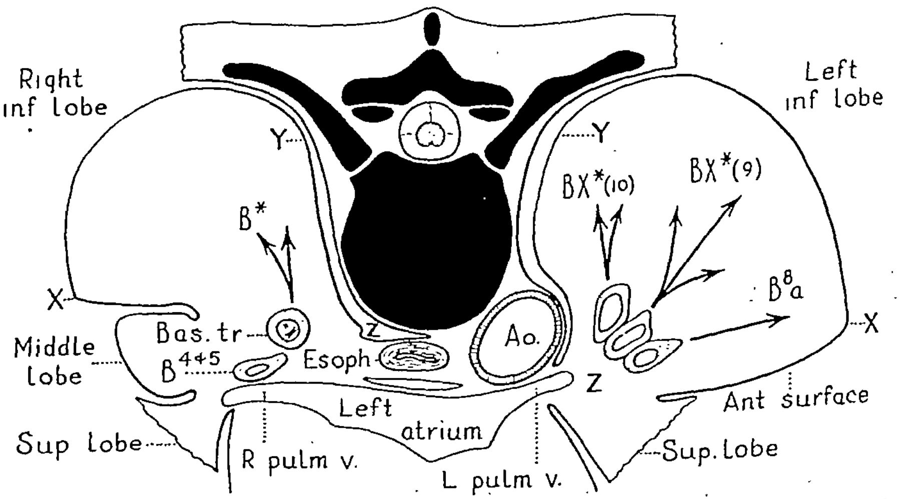
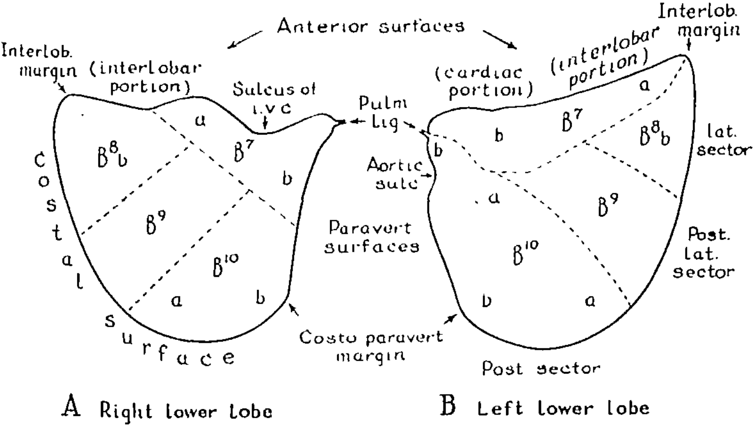


Fig. 3.—Transverse section of the lower lobes of an intact thorax to illustrate the differing direction of the subsuperior bronchi on the two sides. On the right, the subsuperior proper (B^*) distributes primarily to the posterior sector of the costal surface; on the left, it may distribute to the posterior sector (see BX^* from B^{10}), but prevailingly it has a posterolateral distribution (see BX^* from B^9). $BX^*(10)$ and $BX^*(9)$ are left accessory subsuperiors, arising just beneath the left subsuperior proper (B^*), which does not appear *in* this figure. *X*, interlobar margin of lower lobe; *Y*, costoparavertebral margin; *Z*, hilum (or, lower down, the pulmonary ligament).

have defined as the d_2 level. In 5 per cent it originates from the highest position indicated in Fig. 2, *B*, that is, above the origin of $B^{7 \text{ and } 8}$; in the remaining 24 per cent it arises from the common stem of B^{9+10}.

Its position on the circumference of the basal stem is varied. In 12 per cent its orifice is posterior (or slightly posterolateral); in another 12 per cent it is posterolateral; and in 5 per cent it is lateral (or slightly posterolateral). (See Table IV, Item II.) Neil and co-workers (1939) "found it situated dorsally, medially, laterally and twice ventrally." Neil's medial subsuperior is presumably one of the high accessory subsuperiors, $BX^*(10)$, which occasionally arises medially in conjunction with a high paravertebral branch of B^{10} (Fig. 6, *E* and Plate 4, No. 60). Neil's ventral subsuperior could have been a high B^8a with posterolateral branches.



A Right lower lobe B Left lower lobe

Fig. 4.—Diagrams illustrating typical arrangement of basal segments in a cross section of the lower lobes made just above the diaphragm, and viewed from below. *A, right lower lobe* (after Smith and Boyden). Note that the medial basal segment (B^7) is placed athwart the other segments and that the latter overlap each other as postulated by Melnikoff. *B, left lower lobe,* illustrating shift in position of B^7: on this side it lies wholly lateral to the pulmonary ligament, failing to reach the paravertebral surface. That site is now occupied by the posterior basal segment (B^{10}); nevertheless, B^7 is as extensive as on the right. Note that the basal ramus of the anterobasal segment (B^8b) supplies the lateral sector of the costal surface; that the lateral basal segment (B^9) supplies the posterolateral sector; and that the posterior basal (B^{10}) supplies the posterior sector and the paravertebral surface.

This brings out the point that it is often hard to distinguish between a laterally originating B^* (for example, Fig. 7, No. 4) and the lateral ramus of the anterior basal bronchus, B^8a (for example, Fig. 7, No. 41). In the former case, a lateral B^* distributes primarily to the posterolateral sector of the subsuperior zone but it has branches that take over the upper anterolateral zone (BX^8a). Superficially the two specimens look somewhat alike, but when the orifice of B^* is noted (Fig. 7, *D*), the bronchus is seen to arise more posterolaterally, behind B^9.

In Fig. 5, specimen No. 39 is somewhat similar, but here (Fig. 5, *H*) the orifice of the subsuperior bronchus is posterior, that is, behind all basal bronchi.

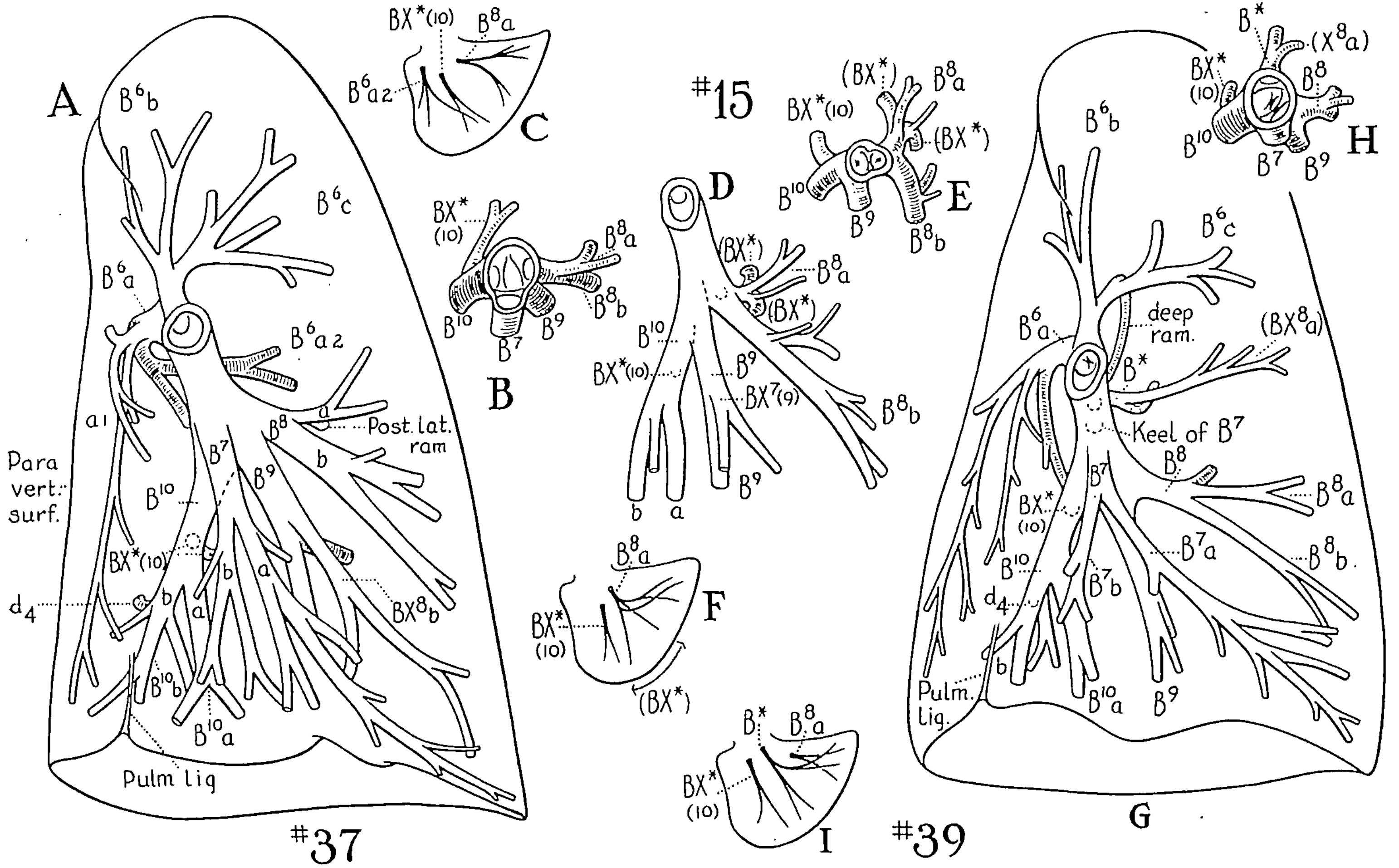


Fig. 5.—Drawings of the bronchial trees of three preserved left lower lobes (X½) to show (1) transitional patterns between B^6a and B^*; (2) anomalous origins of B^7 and B^8. *A, B, C* anterior dissection, orifice of basal trunk, and cross section, respectively, of specimen No. 37. B^8 is typical except for a posterolateral ramus that simulates the distribution of a subsuperior; B^7 arises separately, as in the right lower lobe; B^9 takes over part of the territory of B^8b (see BX^8b). *D, E, F* are corresponding views of specimen No. 15. B^8a is atypical in that it has an accessory subsuperior rami (BX^*). B^7 (as BX^7) arises anomalously from the lateral basal (B^9). *G, H, I* are corresponding views of specimen No. 39. B^* (the subsuperior) takes over part of the territory of the lateral ramus of B^8 (see BX^8a). B^7 again arises separately from B^8.

It is a subsuperior both because of its origin and because of its distribution; yet, again there is a secondary distribution to the B^8a zone.

In specimen No. 15 (Fig. 5) the situation is reversed. Here the corresponding bronchus has a lateral origin in common with B^8b and therefore its main stem must be designated B^8a. The deeper portion of it, which distributes to the subsuperior zone, may be called a displaced subsuperior, $BX^*(8)$. In specimen No. 37, B^8a makes a lesser contribution to the subsuperior zone (see Fig. 5, *C*) and therefore is merely designated B^8a.

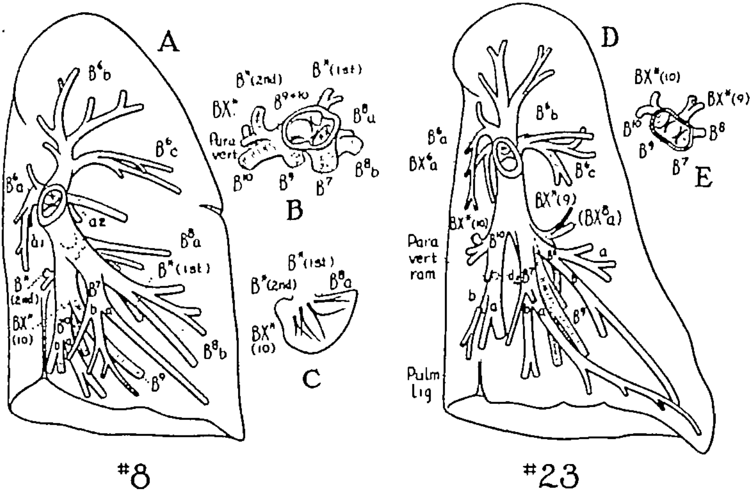


FIG. 6.—Drawings of two preserved specimens (X ⅔) illustrating the prevailing (bifurcate) and atypical (trifurcate) mode of division of the basal trunk. *A, B, C,* are anterior dissection, orifice of basal trunk, and cross section, respectively, of specimen No. 8. The trunk bifurcates into an anterior (B^{7+8}) and a posterior division (B^{9+10}). *D, E,* are anterior dissection and orifice, respectively, of specimen No. 23, showing a trifurcation into three stems, B^{7+8}, B^9, and B^{10}. Incidentally, these figures show how a high division of B^9 and B^{10} may convert a subsuperior proper (B^*, 2nd, of No. 8) into an accessory subsuperior (BX^*[10], of No. 23).

Brock also realized the difficulty of describing these transitional forms. His solution for handling a laterally originating B^* like that in Fig. 7 (No. 4), is to call it a "supernumerary axillary anterior basic bronchus," as if it were the axillary (lateral) ramus of B^8 which had arisen higher than usual—that is, from the main stem. In his own words he preferred "to restrict the name subapical [subsuperior] to the one inconstant but commonest, posterior bronchus and to name and describe any others separately (see axillary branches, p. 63)." However, he apparently did not realize that the one inconstant subsuperior bronchus arising from the lower lobe stem on the left side distributes to the posterolateral sector. Thus, if his preference were followed, most left subsuperiors would have to be called supernumerary axillary middle basals, which would destroy the useful concept of an intermediate zone interpolated between the superior and basal segments. Furthermore, Brock's use of the term axillary to cover bronchi distributing to lateral, posterolateral, and posterior sectors of the lower lobe, while having perhaps a certain clinical value, is not precise enough to be of use in analyzing patterns.

2. *The Accessory Subsuperior, $BX^*(10)$.*—An accessory subsuperior arises as a single branch of the posterior segmental bronchus (B^{10}) in 71 per cent of specimens and as two separate rami in 13 per cent. When single its keel lies typically within a distance of 12 mm. below the keel separating B^9 and B^{10}. When two are present they originate at the levels shown in Fig. 2, *B*, but may distribute to the same surface level (Plate 4, No. 54). In view of the marked lateral shift of B^* on the left side, it is interesting to note that $BX^*(10)$ also distributes to a posterolateral sector in one-half of the specimens in which it occurs (Fig. 5, *C*, No. 37). This is in marked contrast to its behavior on the right side, where it invades the posterolateral sector in only about one-sixth of the cases in which it is the only subsuperior present. The percentages for the left side are given in Table V.

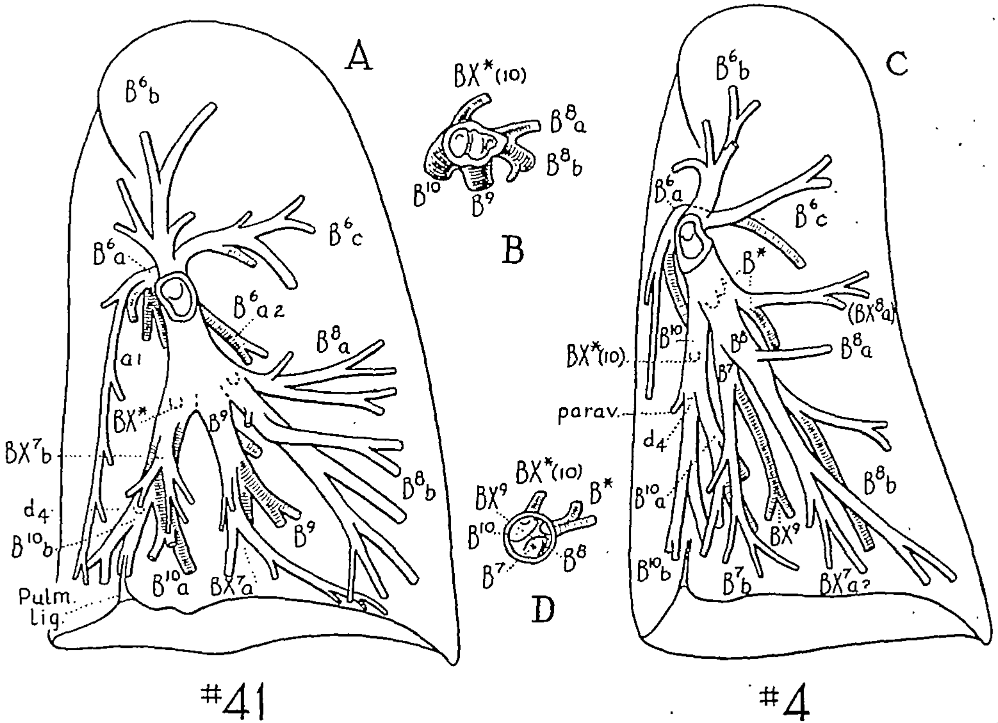


Fig. 7.—Sketches of two preserved specimens (X ½) illustrating bizarre origins of the medial and lateral basal bronchi, B^7 and B^9. *A, B,* are anterior dissection and orifice of basal trunk of No. 41. B^7a (as BX^7a) arises as an accessory branch of B^9. B^7b (as BX^7b) originates as an accessory branch of B^{10}. *C, D,* are corresponding views of specimen No. 4. B^9 (as BX^9) arises as an accessory branch of a deep-lying B^7.

3. *The Accessory Subsuperior $BX^*(9)$.*—An accessory subsuperior arises in 67 per cent of specimens from near the top of the lateral basal segmental bronchus (B^9). Like $BX^*(10)$ its keel lies typically within 12 mm. of the distance below the keel of B^9 and B^{10}. Prevailingly it distributes to the posterolateral sector (see Table VI) and, as would be expected, makes an angle of about

TABLE V. DISTRIBUTION OF BX*(10)

I. Sector	
1. Wholly posterior (Plate 4, No. 60)	41%
2. Posterior and posterolateral (Plate 3, No. 53)	26%
3. Posterolateral only	16%
4. Posterolateral and lateral (Plate 2, No. 58)	1%
II. Angle With Anterior Surface of Lobe	
1. 75-100 degrees (Plate 2, No. 58)	61%
2. 45-50 degrees (Fig. 7, *B*)	23%

45 degrees with the anterior surface in the majority of specimens (see Fig. 3). However, it has a strong tendency to shift laterally. In this respect it resembles the subsuperior proper (see Table IV). It is homologous with B^9a of the right lobe, but on the right side B^9a does not usually participate in the subsuperior zone, since the subsuperior zone of the right lung is primarily restricted to the posterior sector.

TABLE VI. DISTRIBUTION OF BX*(9)

I. Sector	
1. Posterolateral only (Plate 4, No. 54)	40%
2. Posterolateral and lateral (Plate 3, No. 55)	14%
3. Posterior, posterolateral, and lateral	5%
4. Posterior and posterolateral	8%
II. Angle With Anterior Surface of Lobe	
1. 40-60 degrees (Plate 2, No. 56)	45%
2. 20-35 degrees	12%
3. 70-90 degrees (Plate 3, No. 55)	10%

To summarize this sector, it has been pointed out that the criterion for defining the subsuperior zone has been the prevailing distribution of the subsuperior bronchus proper (B*). On the right side, it is prevailingly posterior, and on the left, prevailingly posterolateral. Any additional bronchi that regularly distribute to this zone have therefore been called accessory subsuperiors, notably BX*(10) on both sides and BX*(9) on the left. Certain other extraneous branches that sometimes invade the posterior or lateral sector of this zone—such as the more posterior rami of B^6a, B^9c, and B^8a—might perhaps be included. Indeed, Ewart describes "a central lozenge-shaped bronchial interspace" in the left lower lobe formed by "the lesser posterior horizontal [BX*(10)] . . . together with the external mammary [B^8a] and the midaxillary bronchus [BX*(9)]." However, it seems sufficient to point out that such extraneous branches may invade this zone and to note the criteria by which they may be distinguished from the true subsuperior and the more frequently occurring accessory subsuperiors.

THE BASAL BRONCHI

The subdivision of the basal trunk below the level of the superior bronchus (B^6) is somewhat different on the left side from that on the right. On the left the medial basal segmental bronchus (B^7) arises more anterolaterally and usually in conjunction with the anterior basal (B^8), instead of being the highest and most medial basal bronchus. For this reason various recent authors have considered B^7 and B^8 collectively as a single segmental bronchus. Here, for purposes of description, at least, they will be treated separately.

TABLE VII. MODE OF BRANCHING OF BASAL TRUNK*

I. Basal Trunk Bifurcates			87%
1. Into B^{7+8} and B^{9+10} (Fig. 6, No. 8)		67%	
2. Into bizarre patterns		20%	
(1) B^7 and $B^8 + (B^{9+10})$ †(Fig. 5, No. 37)	8%		
(2) B^{10} and $B^8 + (B^{7+9})$ (Fig. 7, No. 4)	7%		
(3) B^7 and $(B^{8+9}) + B^{10}$ (Fig. 5, No. 39) B^8 and $(B^{9+7}) + B^{10}$ (Fig. 5, No. 15) B^{7+8a} and $B^{8}b + {}^{10}$ (B^9 absent)	5%		
II. Basal Trunk Trifurcates‡			
Into B^{7+8}, B^9, and B^{10} (Fig. 6, No. 23)			13%

*The subsuperiors are ignored in this classification, together with such minor branches as high paravertebral branches of B^{10}.

†Resembles typical pattern of right basal trunk.

‡Counted as a trifurcation when keels were not more than 4 mm. apart. In another 7 per cent the keels were a few millimeters further apart.

Below the point of origin of B^{7+8}, the basal trunk continues as the common stem of the lateral and posterior segmental bronchi (B^9 and B^{10}, respectively). In other words the prevailing pattern, contrary to what has hitherto been supposed, is a bifurcation of the basal trunk into B^{7+8} and B^{9+10} (Fig. 6, No. 8).* Trifurcations occur in only 13 per cent (Fig. 6, No. 23). The percentages and varying combinations are recorded in Table VII.

THE MEDIAL BASAL SEGMENTAL BRONCHUS (B^7)

The medial basal bronchus arises in common with the anterior basal (B^8), or one of its two branches, in 87 per cent of specimens (Table VIII). This short stem emerges from the anterolateral aspect of the basal trunk. Prevailingly (47 per cent), B^7 lies somewhat anterior to B^8 (Fig. 6); frequently (35

TABLE VIII. THE MEDIAL BASAL BRONCHUS (B^7)

I. Origin			87%
1. Arises in common with B^8 (or one of its rami)*			
(1) B^7 lies in plane anterior to B^8	47%	(Fig. 6)†	
(2) B^7 and B^8 lie in same plane	35%	(Plate 1, No. 52)	
(3) B^7 lies deep to B^8	5%	(Fig. 7, No. 4)	
2. Arises separately from B^8 on anterior side of main stem (Fig. 5, No. 37, No. 39)			10%
3. Absent as such but occurs as accessory ramus of B^9 or as rami of B^9 and B^{10} (Fig. 5, No. 15; Fig. 7, No. 41)			3%
II. Distribution			
1. Supplies the whole width of the inferior portion of anterior surface (Plate 3, No. 53)			55%
2. Supplies medial ½ to ⅔ of inferior portion of anterior surface (Plate 2, No. 58)			20%
3. Supplies the whole inferior portion of anterior surface and also a sector of costal surface			17%
(1) By spreading of its branches (Plate 3, No. 55)		10%	
(2) By virtue of accessory rami (BX^9 or BX^8b) (Plate 4, No. 60)		7%	
4. Supplies medial ½ to ⅔ of inferior portion of anterior surface and also a costal sector (BX^9 or BX^8b) (Fig. 7, No. 4)			5%
5. B^7 absent as such; represented by accessory rami of adjacent bronchi (Fig. 7, No. 41)			3%

*In one specimen B^7 was represented in its usual place by only one of its two rami. B^7a; the other ramus, B^7b was displaced, arising as an accessory branch of B^9 (plate 4, No. 54).

†In one specimen B^7 and B^8a arose together, more anteriorly than B^8b.

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*Davis (1929) found a trifurcate pattern in 53 per cent of 32 lungs.

per cent), it lies in the same plane (Plate 1); rarely (5 per cent) it lies deep to B^8 (Fig. 7, No. 4).

Of special interest are the 10 per cent of specimens in which B^7 arises separately from the basal trunk thereby simulating the appearance of its homologue in the right lung. Two such specimens are displayed in Fig. 5 (No. 37 and No. 39). Dévé (1900) was the first to describe this pattern and De Pablo (1940), Hagens (1943), and Foster-Carter (1946) have reported it more recently.

In 3 per cent, B^7 is absent from its usual position, arising as an accessory ramus of B^9 or as rami of B^8 and B^{10} (Fig. 5, No. 15; and Fig. 7, No. 41).

Since the typical pattern in the left lung is characterized by a common origin of B^7 and B^8, many different views have been held with respect to the nature of B^7. Some authors (Aeby, Kramer and Glass, Nelson, Neil, etc.) stated that B^7 is not represented in the left lower lobe. Others (Pierce and Stocking, 1939, Foster-Carter, 1942, Adams and Davenport, 1942, Jackson and Huber, 1943, Brock, 1943, etc.) relegate it to the status of a subsegmental bronchus or less. Only a few investigators, Lucien and Weber (1936), De Pablo (1940), Appleton (1944), etc., maintain that B^7 is a distinct segmental bronchus.

On the basis of its distribution in the sixty specimens presented in this study, we have reluctantly come to the conclusion that it should be considered as a major bronchus. The mere fact that it arises in conjunction with B^8 is no more relevant than the fact that the segmental bronchi of the middle lobe have a common stem.

When the typical distribution of the left medial basal bronchus is compared with the right medial (Fig. 4), it will be noted that the two bronchi aerate an equally large zone. The difference is that on the left B^7 has shifted laterally. Instead of supplying part of the paravertebral surface and part of the anterior surface, it has withdrawn from the paravertebral surface (contrary to Lucien and Weber) and extends from the pulmonary ligament to the interlobar margin. Finally, analysis of the anterior basal bronchus (B^8) shows that it and not B^7 is the defective bronchus (vide infra).

The details of the distribution of B^7 are recorded in Table VIII. In 55 per cent of specimens (Item II: 1) it aerates the whole inferior portion of the anterior surface of the lobe, as measured along the inferior margin (Plate 3, No. 53). In a smaller group (20 per cent: Item II: 2) it supplies only the medial one-half to two-thirds of the lower anterior surface (Plate 2, No. 58). In the remaining 22 per cent in which B^7 is present (Items II: 3 and 4) it invades the costal surface, either by spreading of its branches (Plate 3, No. 55) or by virtue of accessory rami (Plate 4, No. 60).

From another standpoint, it also deserves an independent status—namely, its resectability. Not only is the bronchus available, being visible at the inferior margin of the hilum (Plate 3, No. 55) but in 25 per cent of specimens its medial border (at the pulmonary ligament) is marked by a fissure which extends well into the diaphragmatic surface (Plate 2, No. 56 and Plate 3, No. 55). In addition, there is usually a notch at that point.

Finally, mention should again be made of those specimens in which the left upper lobe is fused to the cardiac surface of the lower lobe. In some instances, B^7 may aerate this adherent laminar portion of the upper lobe (Plate 4, No. 54 and No. 60); in others, the latter is supplied by a lingular bronchus (Plate 2, No. 56).

THE ANTERIOR BASAL SEGMENTAL BRONCHUS (B^8)

The anterior basal segmental bronchus is the least constant of the basal bronchi of the left lower lobe and thus presents a marked contrast to its homologue of the right side; for in 27 per cent of specimens it is defective either in origin—that is, one of its two rami arises as an accessory branch of another bronchus—or it fails to reach the diaphragmatic surface.†

Typically (in 70 per cent of specimens), the anterior basal arises with the medial basal (B^7) through the medium of a short common trunk which may be seen at the lower end of the hilum arising high up on the anterolateral aspect of the basal trunk (Plate 2, No. 58). In another 13 per cent (Table IX, Item I: 2), it arises alone at the site of the common trunk (Fig. 7, No. 41). In the remaining 17 per cent either its basal ramus (B^8b) or its lateral ramus (B^8a) is absent as such, being represented by an accessory branch of another bronchus (Item I: 2, [2] and [3]). In general the accessory lateral rami arise from the subsuperiors, $BX^{*}(9)$ or B^{*} (Plate 1, No. 51), and the accessory basal rami take origin from either the medial or lateral basal bronchi, B^7 and B^9 (Plate 4, No. 54; Plate 3, No. 53). In addition, rami from adjacent bronchi may spread into the territory of B^8, crowding the latter, for example, out of the diaphragmatic surface (Plate 1, No. 52).

TABLE IX. THE ANTERIOR BASAL BRONCHUS (B^8)

I. *Origin*		
1. Arises from a short common trunk with B^7		70%
2. Atypical origin		30%
(1) B^8 arises alone from basal trunk in its usual place† (Fig. 5)	13%	
(2) B^8a only arises with B^7	8.3%	
BX^8b from basal trunk		
BX^8b from B^9 (Plate 3, No. 53)		
BX^8b from B^7 (Plate 4)		
(3) B^8b only arises with B^7	8.3%	
BX^8a from $BX^{*}(9)$		
BX^8a from B^{*} (Plate 1, No. 51)		
II. Distribution		
1. Reaches diaphragmatic surface (Plate 2, No. 56)		83%
2. Fails to reach diaphragmatic surface (Plate 3, No. 55)		17%

†In one specimen B^8 arose in conjunction with B^9.

The normal distribution of B^8 on the anterior surface is best illustrated by Plate 2, No. 56. The lateral ramus (B^8a) supplies the upper half (upper third to two-thirds) of that part of the anterior interlobar surface which lies below the superior segment, together with the adjacent lateral costal surface (Plate 2, No. 58). Since the zone between B^6 and B^8b is at the same level as the interpolated subsuperior segment, the subsuperiors (B^{*}, and BX^{*} from B^9)

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†In 17 per cent, it is defective in number of branches (Table IX, Item I:2), and in 17 per cent it fails to reach the base of the lung (Item II:2). Since seven of the 34 per cent are atypical in both respects, there remains a total of 27 per cent defective segments.

frequently send accessory branches to the B^8a zone (Fig. 6, No. 23) and sometimes take it over completely (Plate 1, No. 51).

The basal ramus (B^8b) typically supplies the lower lateral half (lower third to two-thirds) of the interlobar surface, that is, lateral to a line drawn from the inferior margin of the hilum to the lateral inferior angle of the lobe (Plate 2, No. 56). In only 27 per cent of specimens does it invade the area medial to this line (Plate 2, No. 58). Its typical diaphragmatic distribution is shown in Plate 2, No. 56. As noted previously, it fails to reach the diaphragmatic surface in 17 per cent of specimens.

Resection of this segment would seem to be feasible anatomically, on account of the high origin of B^8 and the accessibility of the segment, but it would seem necessary to anticipate the numerous variations described here.

THE LATERAL BASAL SEGMENTAL BRONCHUS (B^9)

Below the origin of B^{7+8} the stem bronchus continues only a short distance (Fig. 2, *B*) before giving off the lateral segmental bronchus (B^9). On the left side, B^9 and B^{10} lie in virtually the same plane (80 per cent of specimens; Table X, I: 1) but the keel separating the two is so tilted that the orifice of B^9 lies slightly in front of B^{10}.

TABLE X. ORIGIN OF THE LATERAL BASAL BRONCHUS (B^9)

I. Arises From Basal Trunk in Common With B^{10}		90%
1. B^9 and B^{10} lie in same plane (Plate 4)	80%	
2. B^9 lies in a plane anterior to B^{10} (Plate 1, No. 52)	10%	
II. Absent as Such (i.e. displaced)		10%
1. Represented by an accessory ramus of B^7 (Fig. 7, No. 4)	7%	
2. Represented by an accessory ramus of $B^{10}a$	3%	
3. Represented by rami of B^7, B^8, and B^{10}		

Usually within a distance of 12 mm. below the keel, a prevailingly posterolateral ramus is given off in 67 per cent of specimens. This contributes to the subsuperior zone and therefore has already been described as an accessory subsuperior—BX^9(9). It is the homologue of B^9a, the lateral ramus of B^9 on the right side. However, on that side B^9 rarely contributes to the subsuperior zone.

Below this ramus the main stem of B^9 continues toward the diaphragmatic surface supplying the posterolateral sector of both the costal and the diaphragmatic surfaces (Plate 2).

In 10 per cent of specimens, B^9 is absent as such, being represented by displaced rami arising as accessory branches of adjacent bronchi. In this respect it resembles the corresponding bronchus on the right side which is absent in 8 per cent of specimens. In 7 per cent it takes origin from B^7 (see BX^9 in Fig. 7, No. 4). In another 3 per cent it arises from either $B^{10}a$ or by a multiple origin from B^7, B^8, and B^{10}. In contrast to this, the accessory BX^9 of the right lower lobe always arises from B^8. In both lobes, however, the contributing bronchi are on the lateral side. As on the right, B^9 is considered to be too deeply placed to be readily resected.

THE POSTERIOR BASAL SEGMENTAL BRONCHUS (B^{10})

The posterior basal bronchus is the largest of the basal bronchi of the lower lobe and is considered by some embryologists to be a continuation of the paravertebral branch in 30 per cent of specimens. Both B^9 and B^{10} originate too deeply to be considered readily resectable as individual units.

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ALVEOLAR CELL CARCINOMA OF THE LUNG (PULMONARY ADENOMATOSIS, JÁGZIEKTE?)

A Multicentric Tumor of Epithelial Origin

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It is customary to think of a carcinoma as being a local disease, at least of having originated in a small area. It is that conception, of course, which is responsible for the prevalence of surgical attack on the condition. That the conception is correct seems to be borne out by the frequent success in obtaining permanent cures by the surgical removal of the diseased part together with the extensions of the growth from its original site. Regarding primary carcinoma of the lung the generally accepted idea, based on evidence accumulated within recent years, is that this condition not only arises in a bronchus, and is therefore more properly called bronchiogenic carcinoma, but that also like other carcinomas it arises in one spot or area. There can be no doubt that this concept is true for the overwhelming majority of cases of primary carcinoma of the lung.

Yet a few case reports have appeared describing a type of tumor, resembling a carcinoma, which seems to have arisen in the cells lining the alveoli rather than in a bronchus. Moreover, this peculiar tumor seems to have a multicentric origin and thus differs from the common and well-recognized bronchiogenic carcinoma. In this respect it has some resemblance to the primary cancer of the lung in the mouse familiar to the geneticists. Also it resembles the interesting and supposedly virus disease of South African sheep, known as jagziekte.

Several interesting questions arise in connection with this condition. If it is a true carcinoma arising in the alveoli, then the controversy about whether or not the alveoli are lined with epithelium seems apparently to be settled in the affirmative. The similarity to jagziekte raises the question of a possible virus infection in man similar to that in the sheep. Finally the question of the best surgical treatment arises.

It is because of these various considerations that the following case is presented. A special reason for reporting it is that apparently it has the longest follow-up period of any case in which the patient has been subjected to surgical treatment. The result seems to indicate that a two-year period of freedom from a manifestation of symptoms after a lobectomy gives no assurance of complete eradication of the disease, as proposed by Osserman and Neuhof[26] from their experience with a somewhat similar case.

Due to the differences in opinion as to its origin, the particular type of lung tumor characterized by the presence of malignant cells lining the alveoli and maintaining for the most part the general alveolar structure of the pulmonary parenchyma (in the absence of any demonstrable primary lesion elsewhere in the bronchial tree) has been given almost as many different names as the number of authors who have described the lesion. Despite the number of reports in the

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literature now there is still no uniformity of opinion in accepting the tumor as originating either in epithelial or mesenchymal elements, or, indeed in fact, in agreeing that it represents a tumor having a multicentric origin.

It has seemed to us, however, that the preponderance of evidence available at present allows us to consider this particular malignant pulmonary lesion as multicentric in origin and derived from endodermal elements. Consequently, it is permissible to encourage the use of the term "alveolar cell carcinoma," implying as it does the presence of epithelial cells lining the alveoli, malignant changes in which are responsible for the production of the tumor under discussion, and we shall attempt to present evidence offering a basis for this opinion. If a single name can thus be utilized in designating this specific lesion, the recognition that it is a distinct entity will be further encouraged, and clinical, pathologic, and statistical knowledge of this lung tumor gathered on a more certain footing. Future inaccurate tabulation of alveolar cell carcinoma as a form of adenocarcinoma of the lung of bronchiogenic origin can also be prevented by this means.

The historical data, incidence, signs and symptoms, and pathology were fully discussed recently by Ikeda[15] and it is not proposed to enlarge upon these phases of the subject. In brief, the tumor has been reported to occur in two forms, a nodular variety and a diffuse type in which confluence of the tumorous areas suggests a pneumonic picture in the gross. The lesions may be restricted to one lobe of a single lung or involve all lobes of both lungs simultaneously, and they occur within the lung parenchyma or appear on the pleural surface. On gross examination the nodules are commonly discrete with good definition from the surrounding tissue, but they may be irregular in outline or coalesce, this coalescence probably representing the basis for the diffuse form of the disease. The bronchi are not involved except as a result of direct invasion by one of the nodular areas, but metastases do take place to the regional lymph nodes, and occasionally to distant organs. The presence of the tumor in the vicinity of a bronchus may cause atelectatic changes and later inflammatory processes in the adjacent and peripherally situated parenchyma.

Microscopically, the tumor is easily recognized by its characteristic morphology, the malignant cells lining the alveoli, and the general structure of the parenchyma being maintained in most areas. The individual cells tend to be polygonal in shape with rather dark-staining, abundant acidophilic cytoplasm and may occur in multiple layers or in papillary projections within the alveoli, which are often completely filled by these formations of malignant cells. The nuclei are somewhat irregular, often hyperchromatic, and mitoses may be seen in keeping with the obvious neoplastic nature of the cells. An outstanding feature is the general lack of invasion of the supporting framework of the lungs, although occasionally a lymphatic vessel may be seen filled with malignant cells (as might be expected from the occurrence of lymph gland metastases) and in a few of the more malignant cases, the cells are found growing wildly in masses and cords among a loose fibrous tissue stroma. The fundamental feature in the recognition of the tumor is, of course, the very definite reproduction of alveolar structure. It is customary to find many frankly malignant areas that are completely separated as far as gross and histologic evidence is concerned, and occasionally other discrete areas are discovered in which single alveoli may show early hyperplasia of the lining cells of the alveolus, suggesting the beginning of still another focus of the tumor.

In the discussion to follow, this tumor is considered under two headings in accordance with the conflicting views held concerning its origin, (a) the multicentric nature of alveolar cell carcinoma of the lung, and (b) the epithelial origin of alveolar cell carcinoma of the lung.

THE MULTICENTRIC NATURE OF ALVEOLAR CELL CARCINOMA OF THE LUNG

Multiple primary malignancies are no longer the rarity that they were once considered to be, but it is still difficult to accept all the cases reported as actual examples of this condition for the criteria followed in assessing the cases are not always clear. However, the following studies, listed in Table I, seem well substantiated and the figures are included to illustrate the incidence of multiple primary malignancies generally without specific reference to the lung.

TABLE I

	NUMBER OF AUTOPSIES IN MALIGNANT DISEASE	CASES OF MULTIPLE PRIMARY MALIGNANCY	PERCENTAGE OF MULTIPLE MALIGNANCY
Owen, 1921	3,000	143	4.8
Orr, 1930			
(1) Personal cases	1,046	8	0.77
(2) Collected data	9,107	76	0.83
Hanlon, 1931	710	18	2.5
Warren and Gates, 1932	1,078	–	3.7
Schreiner and Wehr, 1934	11,212	307	2.7

The skin and intestinal tract are most commonly affected, with the female breasts and genital organs of both sexes involved with greatest frequency in paired structures. The rarity with which the lung is affected is illustrated in a recent study by Slaughter[20] of 1,868 collected cases of multiple primary malignancy in which he found 749 instances (40 per cent) in the same organs or systems, but not a single case in the lung, and 269 cases (14.4 per cent) in paired organs with only 3 reports of lung involvement. In addition, carcinoma of the lung occurred relatively uncommonly also in association with primary carcinomas affecting organs of other systems, there being in this series only 34 reports of such an association, representing an incidence of only 1.8 per cent as compared to 7 per cent for the stomach, 6 per cent for the prostate, and 4 per cent for the kidney.

Billroth's[4] original view (expressed in 1889) that three conditions must be satisfied before one can consider such lesions as examples of true multiple primary malignancy is now universally considered too exacting. To rule out lymphogenous and hematogenous metastases, extension by continuity or contact, and implantation or transplantation, he suggested that: (a) Each carcinoma must have a different histologic structure, (b) the histogenesis of each carcinoma from a different parent epithelium must be proved, and (c) each carcinoma must form its own metastases.

However, in certain types of multiple carcinomas, generally accepted as primary in origin, these conditions are certainly not fulfilled as, for example, in the case of the occurrence in multiple form of basal cell carcinoma of the skin which rarely metastasizes; for this reason Goetze[11] suggested the necessary criteria might be the following: (a) The macroscopic and microscopic appearance of the tumors must be that of the usual carcinomas of the organs involved, (b) exclusion of metastases must be certain, and (c) diagnosis may be confirmed by the character of the metastases in each case.

If one now accepts on this basis that such lesions as multiple skin carcinomas and multiple malignant polypi of the colon are true multiple primary malignancies, it would seem possible to accept multiple areas of malignancy in the lung as representing multicentric neoplasia, provided that it was possible to satisfy, by meticulous pathologic examination, conditions such as those to be outlined here, which were originally described by Cassili and White[7] in deciding that a case which they reported was in fact an example of multiple lung tumors arising independently from the alveolar lining cells.

1. No primary tumor could be found elsewhere in the body.
2. No primary tumor could be demonstrated in the bronchial tree. In this connection Weller[33] has stated that one must always search until the primary lesion is actually found, because he feels that all such multiple lesions are the result of lymphatic spread.
3. The tumor must be multicentric with areas of uninvolved parenchyma separating the malignant nodules.
4. The neoplastic cellular process must be confined to the alveolar sacs.
5. The interstitial framework of the lungs, except for the lymphatic vessels, must be generally free of the process.
6. The anatomy and histology of the nodules must be unlike any other primary lung process.
   (The fact that all lobes of the lungs were affected in their case seems of relatively little importance.)

Ever since Müller[21] divided the cases in which multiple primary malignancy was present into three groups (1) involving the same system (for example, skin and intestinal tract), (2) involving symmetrical paired organs (for example, breasts, ovary, testis, kidney, lung) and (3) involving distant organs of separate systems, it has been most difficult to satisfy the necessary criteria in the second group (including as it does the breast and genital organs); and it has also been customary to exclude from consideration (as do Warren and Gates[32]) cases of malignancy which is frequently multicentric in origin affecting a single visceral organ, such as carcinoma of the liver, although there seems little doubt that the separate lesions in this case arise independently. As a result of this latter exclusion, similar lesions of the lungs, such as those under consideration in this paper, have also commonly not been included in studies of multiple primary malignancy.

Although multiple lesions of the skin and those occurring in widely separated colonic polypi are now generally regarded as distinct primary changes, it is more difficult to rule out a metastatic basis in the case of paired organs such as the breast and the ovary, where multiple malignancies are next most commonly seen. As mentioned previously, reports in the literature venturing to call these lesions primary in type are often open to criticism. In fact Warren and Gates[32] also exclude most of these cases from their survey.

It is perhaps more difficult still to be certain that multiple lesions in a single lung can be considered to have arisen independently when spread may have occurred along alveolar walls, by the readily available vascular or lymphatic channels or by the intraluminal dissemination of cells with the air motion of ordinary or forced respiration. Nonetheless, sufficient reports have now appeared in which the histologic features are essentially the same regardless of the fact that the change may appear in discrete and widely separated areas or show a tendency to become confluent that it seems unlikely that spread by any of these means could have been responsible, particularly when the conditions outlined according to Casilli and White[7] are satisfied.

The observation that the experimentally produced lesions of alveolar hyperplasia, adenomatosis, and carcinomatosis to be mentioned later are commonly multiple in occurrence and that many of these changes appear simultaneously lends further credence to the view that similar changes occurring spontaneously in the human being are multicentric. In addition, Ikeda[15] stressed the point that in one of the cases he reported each of the nodules actually represented a well-developed intra-alveolar papilloma with a definite central fibrous core and he felt that this further observation also favored the theory of multicentric origin of the tumor.

In summary, then, it becomes apparent that this specific entity which has probably often been labeled an adenocarcinoma in the past is being reported with increasing frequency (for example, Adler[1] 20 cases in 1912, Neuberger and Geever[22] 25 cases in 1942, Ikeda[15] 4 cases in 1945) as its distinctive histologic appearance is accepted, and the typically multiple arrangement of neoplastic areas coupled with the failure to demonstrate a hidden primary tumor permit general acceptance of the view that it has a multicentric origin.

THE EPITHELIAL ORIGIN OF ALVEOLAR CELL CARCINOMA OF THE LUNG

Just as there has been hesitation in accepting the multicentric origin of pulmonary tumors having this distinctive histologic appearance, so also has there been lack of general agreement concerning the presence or absence of a layer of cells lining the alveoli from which this neoplasm might arise. Furthermore, disagreement has been apparent in deciding whether this layer of cells, if it is actually present, develops from endodermal or mesenchymal elements and, consequently, whether it is proper to name the tumor a carcinoma at all.

It is undoubtedly difficult, if not impossible, to demonstrate a lining layer of cells in the alveoli of normal lung tissue, but as Miller[20] points out in his monograph, it is often necessary for the pathologist to turn to normal tissues when in doubt as to the correct designation of a certain cell or group of cells, and in a similar manner the normal histologist may be obliged to inspect pathologic material in order to obtain better insight into the structures under observation. In the case of the lung, he has shown beautifully the presence of a continuous layer of lining cells in the alveoli when they are rendered visible by being separated from the underlying framework of connective tissue and vessels by (a) the pouring out of a serous exudate behind them in a pneumonia or (b) the mechanical edema of mitral stenosis with insufficiency of the valve. In these cases the lining appears as a layer of flattened epithelium in a continuous sheet, the nucleus bulging out in the thinned-out portion of the cell.

Miller[20] also pointed out that the presence of "pores" in the alveolar walls may be explained on the basis of pathologic changes occurring when the epithelium is pushed off in this manner and that they are not necessarily normally present. When the epithelium is pushed off from diametrically opposed sides of an alveolar wall, the serum poured out from the capillaries then finds an exit into adjoining alveoli and with the conversion of fibrinogen into fibrin the characteristic strands of fibrin passing through the alveolar wall are formed (see Macklin).[17] In these cases epithelial cells may be seen attached to the strands of fibrin as they leave the alveolar wall. Macklin[18] demonstrated residual epithelial cells on the pulmonary alveolar walls in various mammals, and he believes it is possible that primary carcinoma of the lung can originate from these cells in man.

Nonetheless, it has been repeatedly observed that cells may be seen lining the alveoli in certain diseased conditions such as chronic passive congestion, lipoid pneumonia, chronic interstitial pneumonia, and other chronic inflammatory processes such as empyema, abscess, bronchiectasis, and tuberculosis. The process appears to be one of hyperplasia of the lining cells and has been termed "epithelization" of the alveoli.

Similar changes which may progress to adenomatous formations and frank malignancy can be produced experimentally by the use of certain chemical carcinogenic compounds. Andervont[2] was able to show for the first time in the lung that carcinogens do not act only locally when he produced primary lung tumors in a certain pure strain of mice by the subcutaneous injection of dibenzanthracene, usually without the occurrence of subcutaneous tumors. Although this strain of mice had a high incidence of spontaneous lung tumors, the induced growth appeared much earlier and it was postulated that following absorption of the carcinogen from the subcutaneous tissue it was carried to the lung where it accelerated the normally occurring carcinogenesis. Lung tumors could, of course, also be produced by the implantation of dibenzanthracene directly into the lungs of this mouse strain.

It is interesting to note that Andervont[3] also bred male and female mice of high pulmonary tumor strain to females and males, respectively, of low pulmonary tumor strain, where all attempts at the induction of lung tumors by dibenzanthracene had failed, and following this females of the first hybrid generation were then mated to their brothers to produce the second hybrid generation. The presence of tumors in a high percentage of both the first (90 per cent) and the second (75 per cent) hybrid generation shows that the susceptibility of the lung to tumor formation induced by the subcutaneous injection of a lard-dibenzanthracene solution is inherited in a dominant manner and is clearly specific for that particular organ.

Multiple adenomatous tumors originating along the alveolar walls have been produced experimentally in mice by Grady and Stewart,[12] and spontaneous multiple tumors of the same type, also in mice, have been observed by Slye, Holmes, and Wells.[30] These adenomatous lesions resemble those described by Cowdry[8] in

South African sheep, a disease known as jagziekte and probably caused by a virus, the counterpart of which has been reported in human beings under the name of adenomatosis by Bell.[5]

We then have definite cellular changes occurring in the alveolar walls in response to many of the factors that have been shown by those working in cancer research to be potentially responsible for tumor formation in general. The factors mentioned have been (a) chronic irritation (chronic inflammations in human beings), (b) chemical carcinogenic compounds (in mice), (c) heredity (in mice), (d) tissue susceptibility (in mice), and finally (e) viruses (in sheep).

Surely, if such responses (hyperplasia or "epithelization" adenomatosis and malignant degeneration) can be regularly induced in the alveolar walls by such varying factors, it would seem that the presence of a cellular lining of the alveoli can no longer be denied, particularly in view of Miller's[20] masterful demonstration of its existence.

However, even if this reasoning is accepted, the question concerning the exact developmental nature of a lesion arising from these cells has still not been answered, for we have yet to decide whether they are of epithelial origin or have a mesenchymal background. No definite answer to this problem seems possible at the present but several factors are highly suggestive.

It is generally accepted by embryologists that the lung develops as a bud of endoderm from the foregut, which branches out into the surrounding mesenchyme as the lungs project into the thoracic celom. On this basis it would seem reasonable to expect that the sacculations (or alveoli) at the end of the branching tree would retain a layer of cells derived from the endodermal bud from which they developed. However, as Barnard and Day[4] pointed out, it has been noted that this cuboidal lining which is admittedly present in early embryonic life seems to disappear from about the fifth month onward and has not been seen to reappear at a later date. Quite possibly in view of Miller's[20] demonstration that a lining does exist, restudy of this problem with more modern techniques, as for example an artificial duplication of the increased venous pressure found in mitral stenosis, might show a similar lining of flattened cells that was not apparent before.

Those who believe that this lining layer does actually disappear theorize that the changes of hyperplasia, adenomatosis, or frank carcinomatosis, as described previously, occur as a result either of hyperplasia of the so-called "septal cells" (Maximow and Bloom)[19] which have a perivascular distribution and are probably mesenchymal in origin, or of extension into the alveoli of cells lining the bronchioles, the epithelial origin of which is uniformly accepted. In the first instance these mesenchymal septal cells may, as Neuberger[23] suggested, gradually form a continuous lining which becomes clearly demarcated from the adjacent tissues, but the absence of reticulum fibrils and the cellular appearance so closely resembling the usual adenomas or carcinomas make this theory appear improbable. It is only fitting to point out, however, that this latter objection is overcome if one accepts the views of Oppenheimer[24] (quoted by Neuberger and Geever) who does not believe that germ layers are specific and believes them capable of undergoing transformation from one type to another under different environmental conditions.

In supporting the second theory Herbut[14] favored the origin of the alveolar cells from the basal cells of the bronchioles since the fact that there is a continuation of these basal cells into the alveolar lining in some cases of bronchiectasis shows that such invasion is possible. Sweany[31] and Jaffe[16] also believe that these basal cells are multipotential and can develop into any type of cell in the bronchial tree including the so-called "alveolar cell," but actually in the literature there does not seem to be any irrefutable histologic evidence that extension of these cells has occurred into the alveoli in the lesions found in this particular type of pulmonary neoplasm.

In this regard Frissel and Knox[10] felt that the alveolar pattern of cellular arrangement merely represents an attempt on the part of the tumor cells to maintain proximity to the available vascular supply in the walls of air sacs and on this basis they felt justified in classifying the lesion as an adenocarcinoma of bronchiogenic origin.

Although it is impossible with our present state of proved knowledge to discard these other theories completely, we believe that there is a sufficient bulk of evidence favoring the presence of a definite cellular alveolar lining to accept its presence and also to consider the cells to be of epithelial rather than mesenchymal origin. Therefore, the term "alveolar cell carcinoma" would appear to be the one most suitable for the future designation of this specific pulmonary neoplasm.

CASE REPORT

The patient, E. L. W., a 35-year-old used car dealer, was admitted to Barnes Hospital on June 1, 1943, at which time he stated that he had suffered from a chronic cough for the past eight to twelve months, which had become productive in the six months prior to admission. At this time he was coughing up approximately one-half cup of foul-smelling, greenish yellow sputum each day, and although there had been no associated pain, voice changes, general weakness, or weight loss, he stated that he had noted a small amount of blood streaking of the sputum ten days before admission. This was the only occasion on which hemoptysis had been present.

There had been some dyspnea on exertion, associated with palpitation and vertigo, and these "dizzy spells" had become sufficiently severe that the patient had consulted a physician concerning them about six months before he was admitted and had been treated for "heart trouble" and "allergy" without any improvement. One month before admission he had been seen by a chest specialist who ordered a chest plate and subsequently referred the patient directly to a hospital.

The past history apart from some mild chronic sinusitis of many years' standing and a "touch of dropsy" did not appear to be significant, nor did the family history or the general functional inquiry. He did not smoke.

Physical examination at the time of admission revealed a few large moist râles at the lung bases posteriorly, with some questionable diminution in the audibility of the breath sounds over the right lung field. The temperature, pulse, respiration, blood pressure, and laboratory findings were within normal limits and the sputum was negative for tubercle bacilli or fungi. There were no other significant findings on physical examination.

A flat plate of the chest showed an infiltrative process in the right lower lung field (Fig. 1) which was nonspecific, and a lipiodol bronchogram showed all the bronchi on the left side as well as those to the right upper and middle lobes to be normal. Only a portion of the right lower lobe bronchus was filled, although several flecks of opaque material were visualized overlying the large area of infiltration.

Diagnostic bronchoscopy was similarly of little help. The trachea contained a little pus but the carina was thin and normal in appearance and the only noteworthy finding in any of the bronchi visualized was a slight bulging in one of the medial divisions of the right lower lobe bronchus from which a biopsy was taken. The pathologic report on the specimen, however, showed no areas suspicious of malignancy and was labeled as chronic inflammation. The material consisted of a polypoid mass covered on all sides with epithelium that occasionally seemed to be undergoing squamous metaplasia. There was a core of fibrous tissue with many blood vessels and a considerable inflammatory process throughout although most of the cells were lymphocytes.

It was felt that an exploratory thoracotomy should be performed and this was done (by E. A. G.) on June 8, 1943.

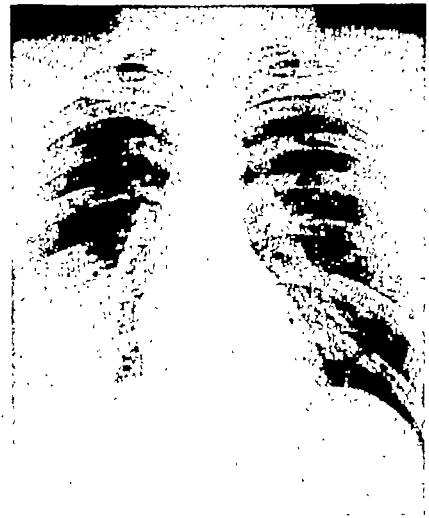

Fig. 1.—Roentgenogram of chest June 2, 1943, just prior to first operation. Homogeneous poorly delineated shadow in right lower chest. Upper lung field clear.

Operation.—On opening the chest there was no evidence of either pleural effusion or adhesions, but there was a mass of fairly firm tissue involving the greater portion of the right lower lobe and extending to the periphery where it caused a very marked dimpling of the visceral pleural somewhat suggesting carcinoma. However, it was felt that the consistency was not sufficiently firm to substantiate this suggestion and it was finally decided that the lesion was inflammatory in nature and consequently a lobectomy was performed. Ordinarily a frozen section would have been made at the time of operation, but the limited facilities for carrying it out because of war conditions made the procedure impossible.

A considerable amount of bloodstained fluid collected in the immediate postoperative period, and as closure had been effected without drainage, it was necessary to perform several thoracenteses to prevent a shift of the mediastinal structures. No evidence of infection appeared, however, and thereafter the postoperative course was uneventful and recovery satisfactory.

Pathologic Report.—The gross appearance was much as described previously with a depressed puckered looking area overlying the actual lesion, which on sectioning was found to comprise a well-encapsulated area of edematous and probably infected tissue. At one edge of this area was a small cavity containing some necrotic tissue, and surrounding this cavity the induration was sufficient to re-arouse the suspicion of carcinoma. It also suggested an organizing pneumonia (Fig. 2).

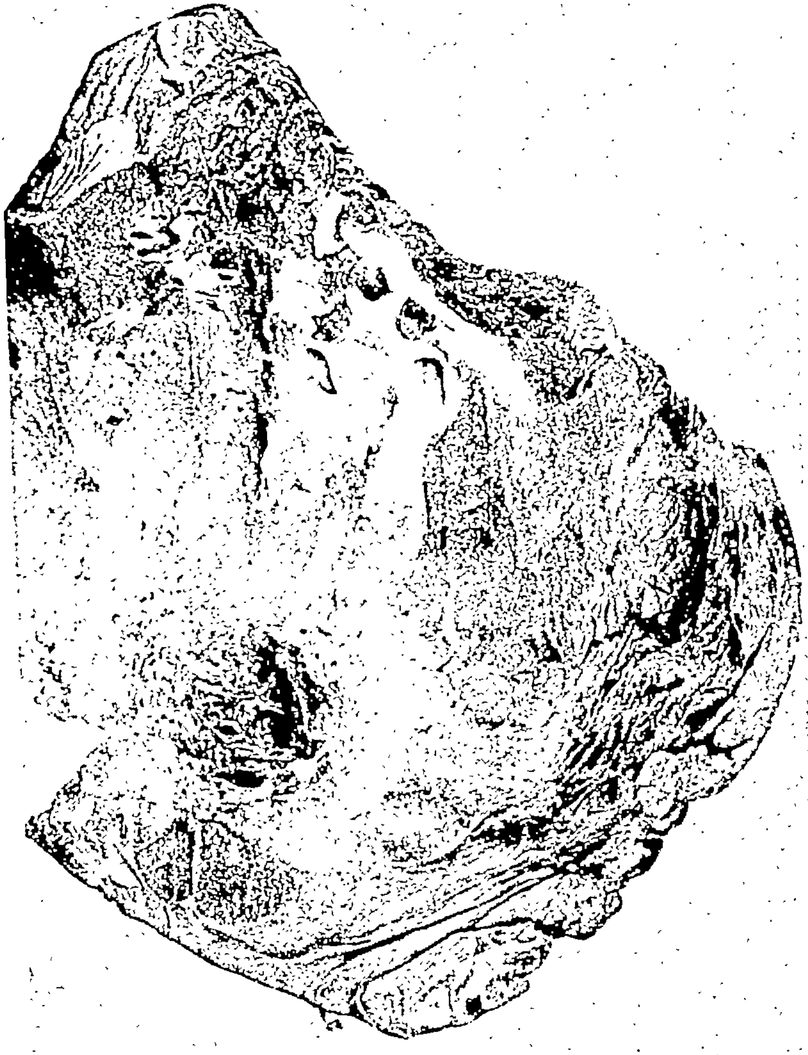

Fig. 2.—Gross specimen of right lower lobe removed June 8, 1943. Note gray-white area which suggests pneumonic process surrounding zone of cavitation. No lymph nodes involved.

Microscopically the tendency to reproduce the alveolar structure was by far the predominating feature of the lesion. Several well-defined areas of lung tissue showed almost complete replacement of the alveolar linings by hyperplastic epithelium comprised of polygonal cells having abundant acidophilic cytoplasm and dark-staining nuclei (Figs. 3 and 4). At the periphery of the lesion there were occasional isolated groups of alveoli in which the epithelium looked hyperplastic with increase in the cellularity, the cells tending to grow in small papillomatous formations projecting into the lumen of the alveoli (Fig. 5). In still other areas the tendency to the reproduction of alveolar structure was not seen, the cells growing wildly in clumps and cords in a loose fibrous tissue stroma. In these zones the malignant cells showed marked variation in size, shape, and staining reaction, and mitotic figures were fairly frequent. The remaining lung parenchyma showed a peculiar lobular distribution of edema and there was a marked chronic inflammatory reaction surrounding all the larger bronchi and bronchioles. There was no evidence either at operation or pathologically of involvement of the regional lymph nodes.

Although there was obviously a great deal of secondary infection and inflammation in the lobe it was recognized at the time by Dr. Nathan Womack that a carcinomatous process appeared to be arising simultaneously in several different regions. Also he called attention to the similarity of this case to jagziekte. Because of this fact and the lack of any evidence of regional metastases, it was decided unwise to consider completion of the pneumonectomy due to the probability that similar areas of involvement were present in the left lung. Perhaps this was an error in judgment in view of the subsequent history.

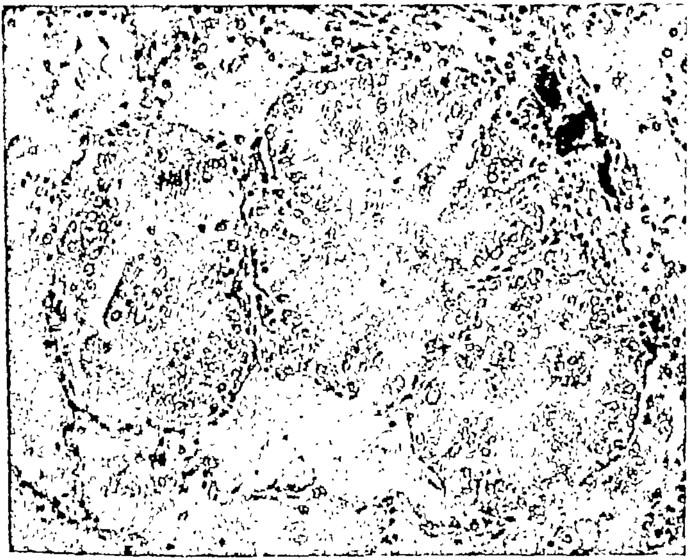

Fig. 5.—Characteristic nodule of well-differentiated tumor with preservation of alveolar pattern surrounded by normal lung parenchyma, first operation. (Photomicrograph ×420.)

Subsequent Course.—The patient made a satisfactory recovery and was well until the fall of 1946. He saw Dr. A. S. Ashmore of Wichita, Kan., in October, 1946, and complained at that time of a productive cough. An x-ray of the chest showed a lesion overlying the diaphragm measuring 2½ inches in diameter. He refused treatment at that time. He began to lose weight and developed pain in the right upper thorax and shoulder girdle region which interfered with the function of the right arm. On July 17, 1947, bronchoscopy was negative and the lipiodol examination was unsatisfactory, but the x-ray demonstrated a dense shadow obscuring the right lower lung field (Fig. 6). In December, 1947, exploration was done at Wichita by Dr. Ashmore and the remaining portion of the right lung resected. At the time of the exploration, tumor resembling "caviar" or "raspberry jelly" was seen growing over the chest wall where it had invaded over an area of 4 inches in diameter. The mediastinal lymph nodes were apparently negative. He made another uneventful recovery from this operation but because of the evidence of invasion of local tissues, he was given intensive postoperative irradiation therapy (7,000 r. through four portals). He was last seen in September, 1948. Bronchoscopy was negative and there were no tumor cells found in the smears from the bronchus. The opposite lung was clear to radiographic examination. He was in

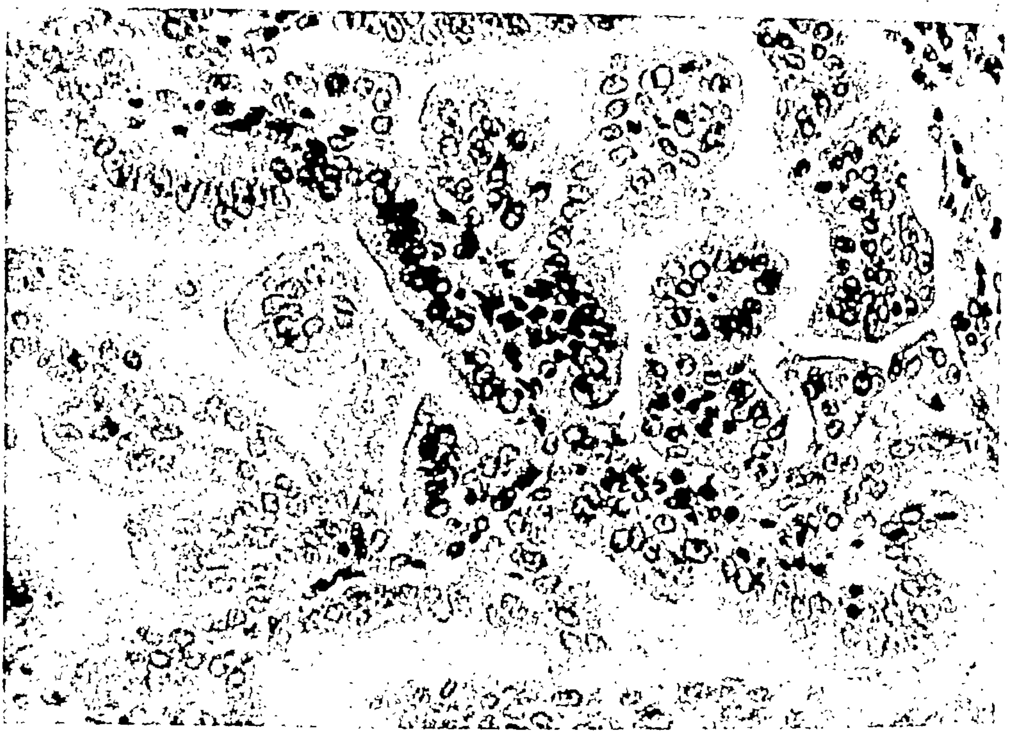

Fig. 4.—Detailed view of nodule of tumor demonstrating its well-differentiated character, first operation. (Photomicrograph ×420.)

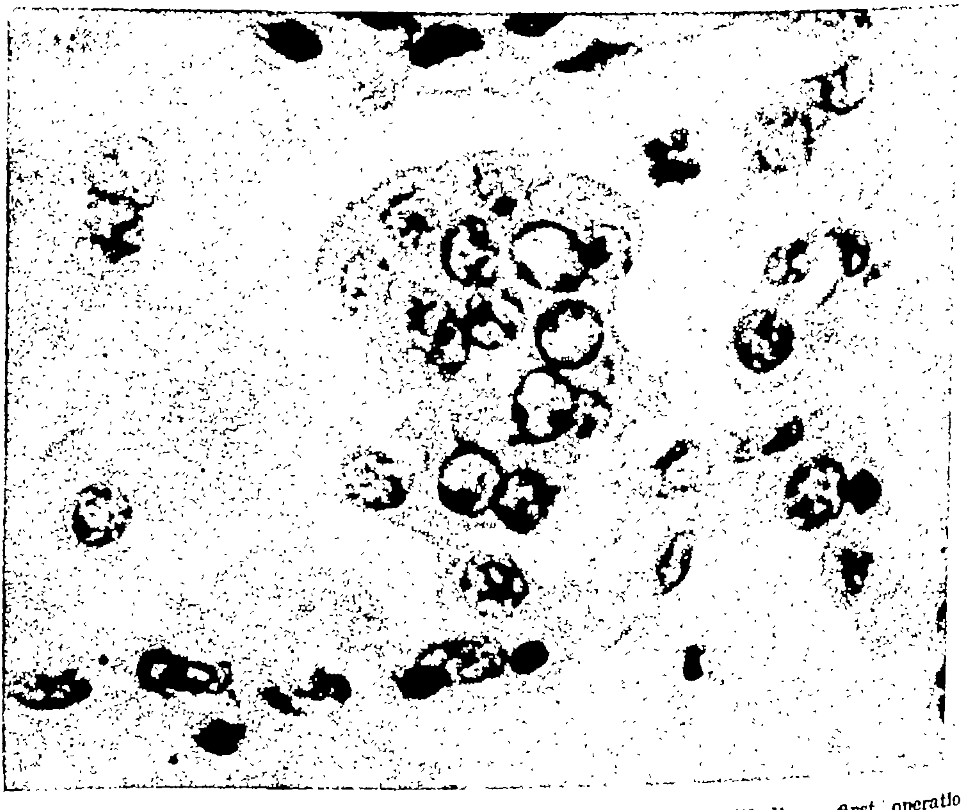

Fig. 5.—Cluster of tumor cells suggesting origin from alveolar epithelium, first operation. (Photomicrograph ×650.)

excellent clinical condition. The microscopic pattern of the tumor found at the second operation was very similar to that seen at the first operation (Fig. 7).*

DISCUSSION OF CASE

This patient has a known duration of his disease of at least six years. The initial lobectomy resulted in freedom from symptoms for about four years. At the time of the second operation the microscopic appearance of the tumor had not changed (Fig. 7). However, there was evidence of local invasion of the chest wall. The regional lymph nodes have remained free from disease. The opposite lung radiographically has shown no change but it is entirely possible that already in this lung there may be the early changes of hyperplasia (epithelization) or benign adenomatosis that are presumably of a precancerous nature.

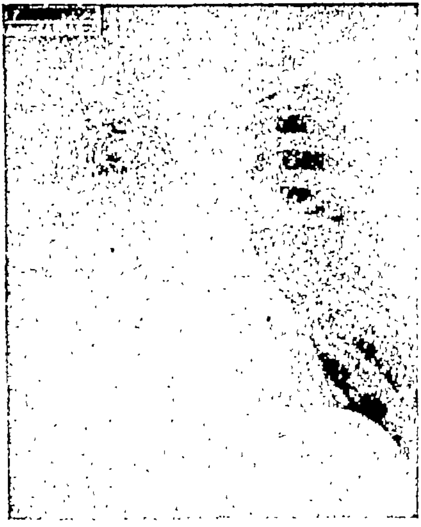

Fig. 6.—Roentgenogram of chest July 8, 1947, just prior to second operation. Dense shadow in right lower chest, high diaphragm, thickened pleura. Left lung clear.

The surgical treatment of this case has resulted at least in very worth-while palliation. The peculiar nature of the tumor, although probably malignant, and the absence of any known surgical experience with it prior to our operation in 1943, made the necessity of total pneumonectomy seem questionable at that time. Perhaps, however, further experience will show that the total removal of the lung is indicated as a palliative procedure in spite of the existing possibility of lesions in the other lung. This case would seem to suggest that. This patient is apparently the third treated surgically and the only one with a long follow-up (Drymalski[9] and Wood and Pierson[34]). Wood's patient had a right lower lobectomy for what was thought to be an inflammatory lesion. A roentgenogram six weeks after operation appeared to be clear of disease. The patient died six months later of carcinoma of the uterus. At post-mortem examination multiple foci of pulmonary adenomatosis were found in all remaining lobes. As Wood's case is very similar to the one reported here in both its clinical course and pathology, it lends further support to the concept of multiple foci of origin. His case also suggests that the case reported here may very well have other foci of tumor in the remaining left lung. Wood recognized the similarity of his case to jagziekte. No doubt the lobar distribution of the signs and symptoms of some of these cases is due to the strategic location of nodules of tumor close to a bronchus. This results in infection distal to the block and these changes give a clinical picture suggesting inflammation. If there is no surgical interference the patient dies of inflammatory complications. The case reported demonstrates the value of surgical resection in the presence of such localized inflammatory complications.

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*We are indebted to Dr. Ashmore for the history of the subsequent course, the x-ray film, and the microscopic sections from the second operation.

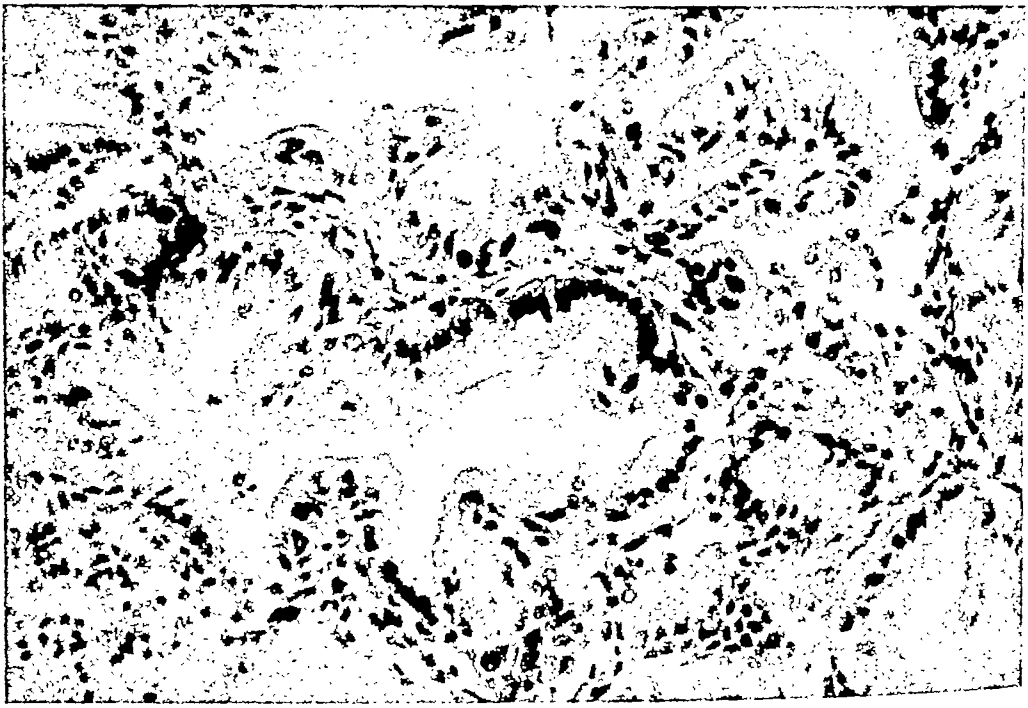

Fig. 7.—Persistent tumor in upper lobe. Note well-differentiated character and similarity to original tumor, second operation. (Photomicrograph ×60.)

SUMMARY

A brief review has been presented dealing with a specific type of pulmonary neoplasm which is characterized by a reproduction of the alveolar structure of the lung. The evidence is discussed which favors recognition of the multicentric nature of the lesion and seems to indicate that it may quite properly be called an "alveolar cell carcinoma." An illustrative typical case is reported which demonstrates the slow growth of the neoplasm and the palliation which may result from the removal of the diseased tissue, although the manifestations of the disease may reappear several years later in a different part of the lungs, as in this case. Also, attention is called to the similarity of this case to jagziekte, the South African disease of sheep.*

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*The pathology of this case has been reviewed by Dr. Lauren V. Ackerman of the Section of Surgical Pathology, Washington University School of Medicine, St. Louis, Mo.

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ECTOPIA BRONCHI

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DEVELOPMENTAL abnormalities of the tracheobronchial tree and of the pulmonary parenchyma are rarely found in life or at necropsy. Even after careful clinical scrutiny one commonly confuses such abnormalities with other pulmonary diseases, or else arrives at a diagnostic impasse. We are principally interested here with ectopia of the bronchi to the left upper lobe and with the resultant varied parenchymal changes in the left lung.

EMBRYOLOGY

Arey[1] in discussing the embryology of the lung stated that in the 3 mm. human embryo the lung first appears as a groovelike evagination on the ventral surface of the esophagus. The enlarged caudad end of this evagination gives origin to two small lung buds. At a later stage, the predestined anlagen of the esophagus and trachea become separated by a constriction. This constriction is separated at the cephalic end by the larynx which is demonstrable at the end of the fifth week in the embryo. Hollow evaginations of the lung buds extend into the surrounding mesenchyme, enlarge rapidly, and branch to produce a treelike tubular system. The surrounding mesenchyme by the end of the seventh week gives rise to muscle fibers, cartilaginous rings, and probably to the lining cells of the alveoli.

Ekehorn[2] and Flint[3] related that the lower, blind end of the primordial trachea becomes fixed, divides, and then projects into each thorax (stem bronchi) where, by a process of arborization and downward growth, the bronchial tree is developed. Heiss[4] believed that a process of infolding and septum formation are the developmental mechanics responsible for the progressive differentiation of the lung. He rejected the term and conception of stem bronchi. For him, stem bronchi are the periphery of the lung from which the connecting and terminal structures of the lung parenchyma are formed by a continuous infolding.

Ferguson and Neuhauser[5] added that as a result of the sequence of embryologic events "complete absence of the lung, with no lung bud or main stem bronchus whatever, indicates a developmental defect which has occurred very early in fetal life."

Gruenfeld and Gray[6] speculated that developmental anomalies of the lung are caused by "phylogenetic newness of the organ" and by the "enormous growth it must undergo in the embryo before its functional form is achieved." They first identify the division of the left stem bronchus into its two main bronchi in the 7 mm. length human embryo. It is interesting that in their review of lung malformations there is not included an example of the type here presented.

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Schneider[7] classified aplastic lungs according to the presence or absence of a rudimentary bronchus or of an aplastic lung. He further differentiated such aplasia into high and low degrees depending on the absence in whole or in part of the bronchial or pulmonary element.

Jordan[8] in his detailed classification of pulmonary anomalies would include the case herein reported as "agenesis of bronchus—to a normal lobe."

Caffey[9] in describing the congenital defects associated with total aplasia of the lung included ". . . . diaphragmatic defects, cardiac and renal malformations, skeletal abnormalities, and absence of the vagus nerve." He considered that the high frequency of association of pulmonary aplasia with hemivertebra justifies considering the combination as a "syndrome." He presented a case showing bronchographic evidence of absence of the eparterial bronchus in association with an hemivertebra of the eleventh thoracic vertebra. Peeler and Phillips[10] cited the finding in a 2-month-old infant of a hypoplastic bronchus to the left upper lobe; however, there was no histologic examination. Cassinelli[11] and Nelson[12] each discussed a similar case which on microscopic examination was found to have a marked diminution in the number of cartilaginous rings. Morelli[13] discussed the possibility that an aberrant pulmonary artery might produce this latter aberration. He, too, cited a case in which the bronchus to the left upper lobe was small and lacking in cartilaginous rings and in total cartilage in the rings present. Diagnosis was made on his patient at 1 month of age, and substantiated at necropsy at 4 months of age. He indicted the aplasia of bronchus and bronchial cartilage as the agent precipitating the emphysema.

In concluding this review of the literature on the embryology of the trachea, bronchi, and lungs, we feel with Harris and Lewis[14] that knowledge of the comparative anatomy and embryology of the bronchial system has not kept pace with thoracic surgery. This lack of information is reflected in the difficult recognition of such lesions.

CASE REPORT

A white boy, aged 9 years, was admitted* April 23, 1947, to Children's Hospital, Akron, Ohio. The child's physical and mental growth and activities were not unusual until 1945, when for the first time he was noted to tire readily and to become dyspneic upon exertion. A physician made the diagnosis of dextracardia and cautioned the parents to limit the child's physical activities; as a result of this the child became acutely aware of his heart and selectively limited his activities to his own favorite sports. During the next two years his only complaint was of shortness of breath; there was no cough, expectoration, chest pain, or known evening hyperthermia.

Physical examination on admission revealed a thin, 9-year-old, intelligent, cooperative white male child in whom positive findings were limited to the thorax. The thoracic cage was asthenic in type with a widening of the intercostal spaces on the right and a narrowing and relative immobility of those on the left; there was a slight list of the left shoulder. The heart rate was 110 with a normal rhythm; the left border of cardiac dullness was retrosternal, the right was at the right nipple. On the left side there was increased resonance and diminished breath sounds and voice conduction. On the right side breath sounds were normal. There was a mild secondary anemia. Intermittent afternoon fever as high as 100.4°

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*On the medical service of Dr. Noah Miller.

Fig. 1.

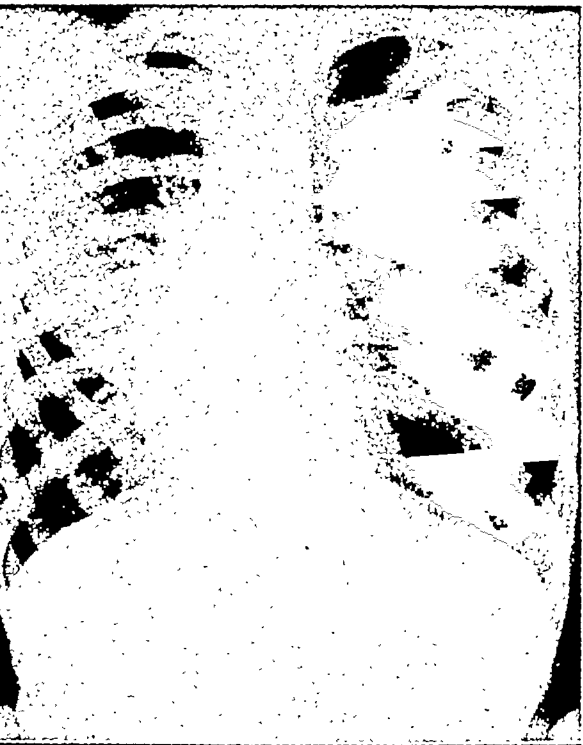
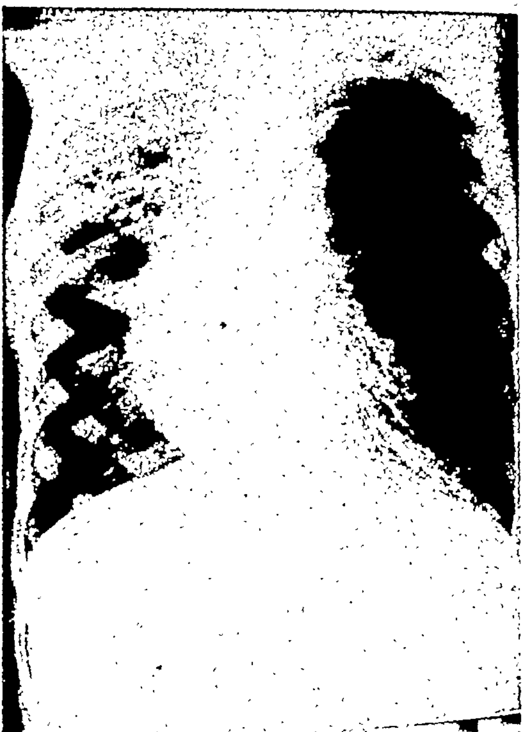


Fig. 2.



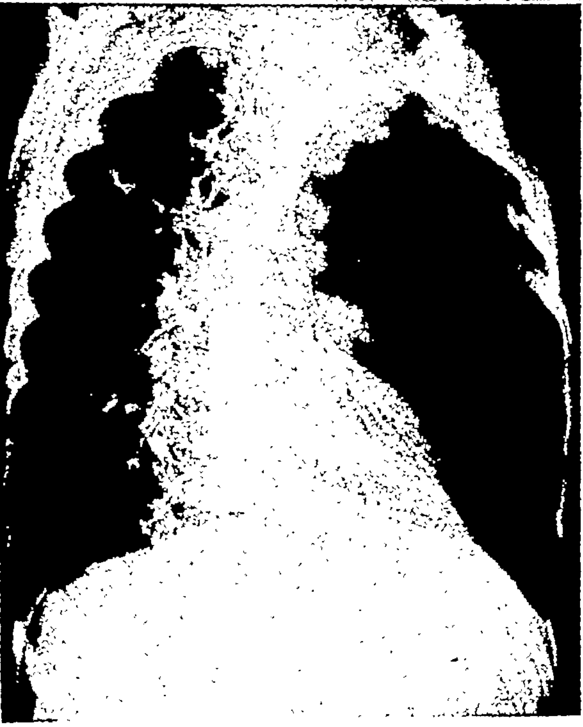


Fig. 3.

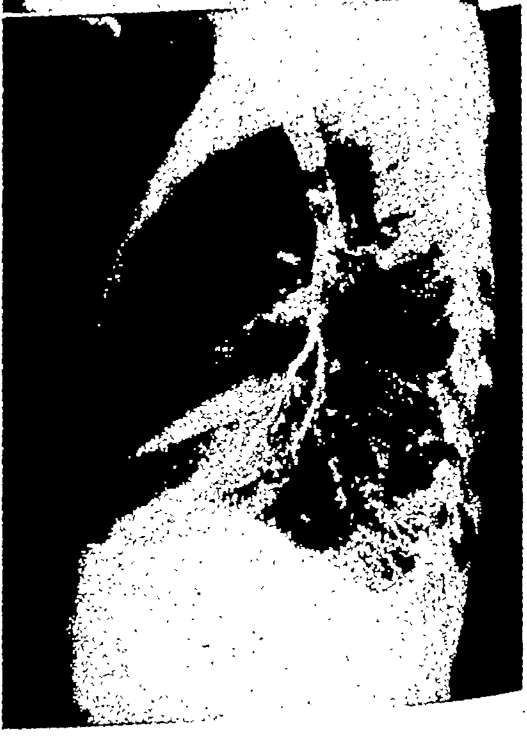


Fig. 4.

Fig. 1.—Anteroposterior roentgenogram showing cardiac shift, radiolucent upper left chest, and small paramediastinal cyst.

Fig. 2.—Anteroposterior bronchogram showing concentration of bronchi in lower thorax.

Fig. 3.—Left postero-oblique bronchogram showing upper lobe bronchi in detail.

Fig. 4.—Left lateral bronchogram showing posterosuperior upper lobe bronchi extending into emphysematous mass.

F. was noted. Intrathoracic pressure readings in the second left interspace were: on inhalation, plus 2 cm. water pressure, on exhalation, plus 6 cm. After aspiration of 200 c.c. of air the pressure readings were: on inhalation, minus 10 cm. water pressure, on exhalation, minus 4 cm. The patient coughed vigorously twice, and following this the readings returned to the pre-aspiration levels.

Anteroposterior x-ray view revealed (Fig. 1) a shift of the mediastinum to the right with a semblance of a dextracardia. A few fine pulmonary markings appeared to radiate from the left hilum into the superior and lateral aspect of the left lung area; these markings radiated from an area 3 cm. in diameter of increased density located just to the left of the hilus. A concentration of pulmonary markings in the lower fifth of the left thorax suggested a compression type of atelectasis. Bronchograms (Figs. 2, 3, and 4) revealed a concentration of an entire normal left bronchial tree in the atelectatic lower lung field, and a composite bronchodiagram, left lateral view (Fig. 5), identifies each bronchus according to the systems of Boyden[15] and Jackson and Huber.[16] In the anteroposterior (Fig. 2)

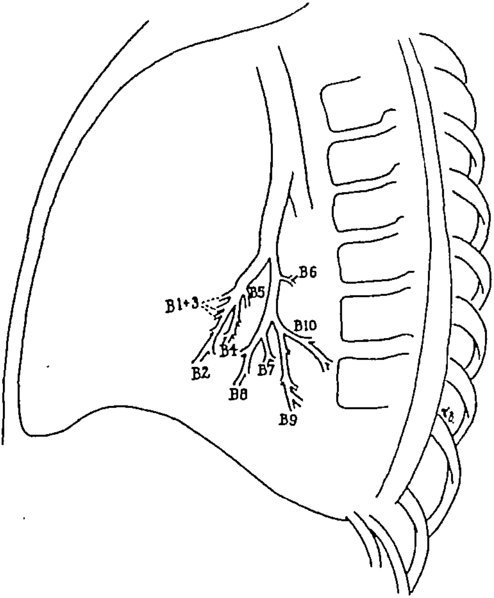


Fig. 5.—Composite bronchodiagram, left lateral, developed from Fig. 4, with identification of bronchi according to the system of Boyden,[15] and correlation (below) to the system of Jackson and Huber.[16]

	UPPER LOBE
BOYDEN	JACKSON AND HUBER
B 1 plus 3	Apical-posterior
B 2	Anterior
B 4	Superior (lingular)
B 5	Inferior (lingular)
	LOWER LOBE
B 6	Superior
B 7 B 8	Anterior-medial basal
B 9	Lateral basal
B 10	Posterior basal

and left lateral (Fig. 4) views there are several fine branches extending cephalad from the first 2 cm. of the upper of the two stem branches of the left main bronchus. These left upper lobe branches are identified (Fig. 5) as Bronchi 1 and 3 by the system of Boyden[15] and Scannell[17] or the posterosuperior segmental bronchi by Jackson and Huber.[16] It was subsequently determined that these bronchi were the only sources of air into the emphysematous upper lobe. The normal complement of upper lobe bronchi readily can be identified in the left posterior oblique view (Fig. 3), and the two bronchi to the lingula are seen to advantage in the lateral view (Fig. 4) to arise as separate branches from the lobe bronchus.

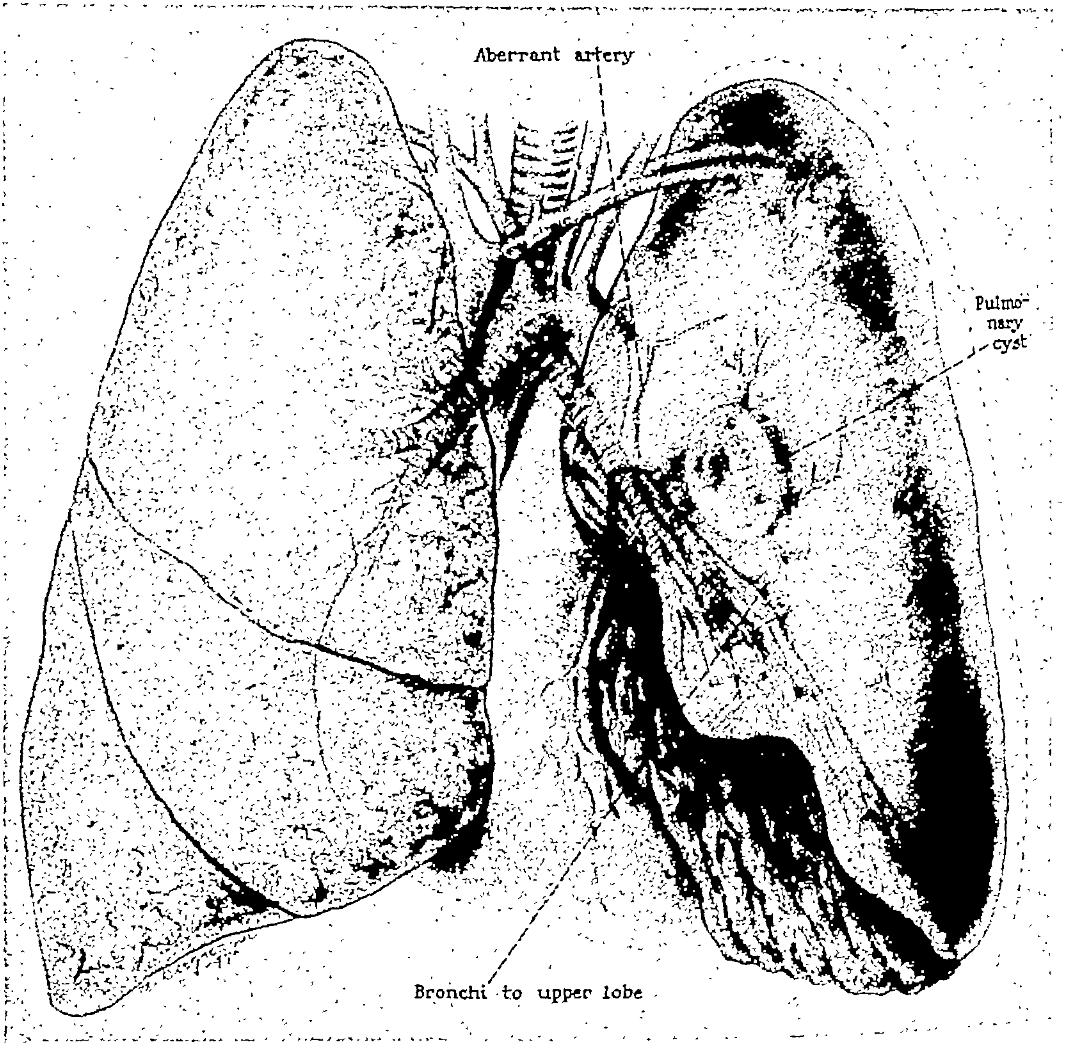


Fig. 6.—Illustrating atelectasis of left lower lobe and mediastinal shift secondary to emphysematous left upper lobe. Note aberrant artery and bronchi to upper lobe.

A diagnosis was made of pulmonary cyst of the left upper lobe with bronchopleural fistula and compression atelectasis of both lobes. It was felt that this cyst was sustained by a "ball-valve mechanism" in the communicating bronchi, and thoracotomy was undertaken in an effort to resect the cyst.

On May 21, 1947, under oral-intratracheal ether-oxygen anesthesia through a posterolateral incision with resection of the fifth rib and section of the fourth and sixth at the tubercle, the left thoracic cavity was opened. A minimal pneumothorax under slight positive pressure was found. The left upper lobe (Fig. 6) filled 80 per cent of the left thorax and obviously was exerting pressure on the atelectatic, dark, fibrotic lower lobe. Re-expansion of this lower lobe was impossible even with 20 cm. pressure exerted through the intratracheal tube. The upper lobe was pink and though spongy in consistency it still was tense and remained so even after the manipulation attendant on pneumonectomy. Increasing the intratracheal pressure increased slightly the tenseness of this upper lobe. The surface of the lower lobe had intercommunicating lobule markings strongly outlined with anthracotic pigment; however, these markings in the upper lobe were much more widely separated and were a bright pink in color. The heart and mediastinum appeared normal. In the hilus of the lung there were several soft lymph nodes measuring up to 1 cm. in diameter; the pulmonary artery and veins were normal in size and position. An aberrant artery 3 mm. in diameter originated from the aorta near the origin of the second left intercostal artery and was directed into the mediastinal aspect of the upper lobe. Total pneumonectomy was performed by individual suture-ligation silk technique, and the thorax was closed without drainage. Convalescence was uneventful, and the patient was discharged on the twelfth postoperative day; a chest roentgenogram taken twelve weeks later (Fig. 7) revealed a satisfactory position of the heart and mediastinum. The patient returned to school where he was found to have a much greater exercise tolerance; the "cardiac consciousness" was gone.

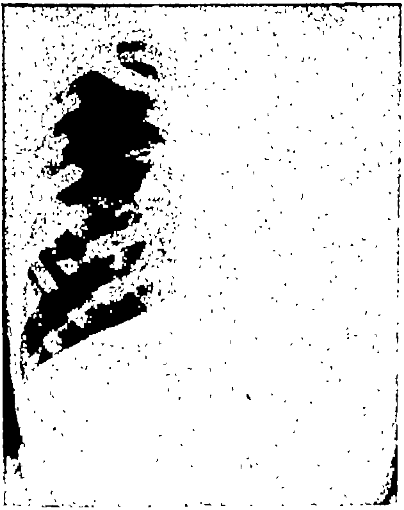


Fig. 7.—Anteroposterior roentgenogram taken twelve weeks postoperatively

Pathology.—The lung measured 6 by 12 by 17 cm.; the pink, emphysematous upper lobe was 5 by 12 by 13 cm. and had the form of a normal but greatly distended lobe. The pulmonary veins were normal in distribution. The pulmonary artery contributed several fibrinous branches to the upper lobe and then ramified throughout the lower lobe. The aberrant artery from the aorta branched soon after entering the upper lobe. The left stem bronchus entered only the lower lobe where it divided into two main trunks (Fig. 3). Just beyond this bifurcation the cephalad of these bronchi gave off three bronchi measuring about 2 mm. in diameter; these were identified as Bronchi 1 and 3 on the bronchodiagram. They extended across the interlobar fissure for about 2 cm. into the substance of the upper lobe where they terminated, patent, in the emphysematous tissue of the lobe. These were the only communications of the tracheobronchial tree with the upper lobe.

Cut section of the upper lobe revealed thin-walled emphysematous blebs throughout, 5 to 10 mm. in diameter. On microscopic examination (Fig. 8) the presence of such tissue was confirmed, and a diminished number of terminal bronchi were seen. Except for the previously described bronchi entering the lobe from the lower lobe there were seen but four or five bronchi, 2 to 3 mm. in diameter, and these radiated into the periphery of the lobe from a hilar cyst 1 by 1 by 2 cm. (Figs. 1 and 6). The cyst wall was firm, 3 mm. thick, and contained gray, inspissated mucoid material; microscopic examination of its wall (Fig. 9) revealed the structure of a large bronchus.

The lower lobe was atelectatic (Fig. 10), and cut section revealed an inordinate number of closely crowded bronchi. Detailed examination of the distribution of these bronchi confirmed the presence and arrangement of bronchi as represented on the composite bronchodiagram (Fig. 5).

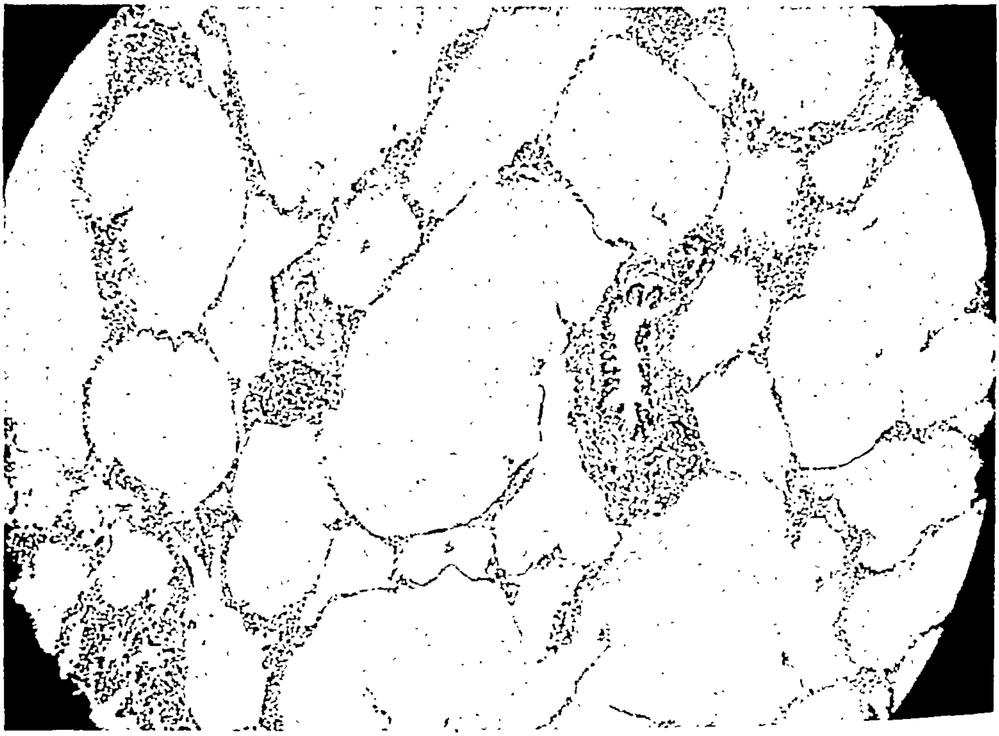
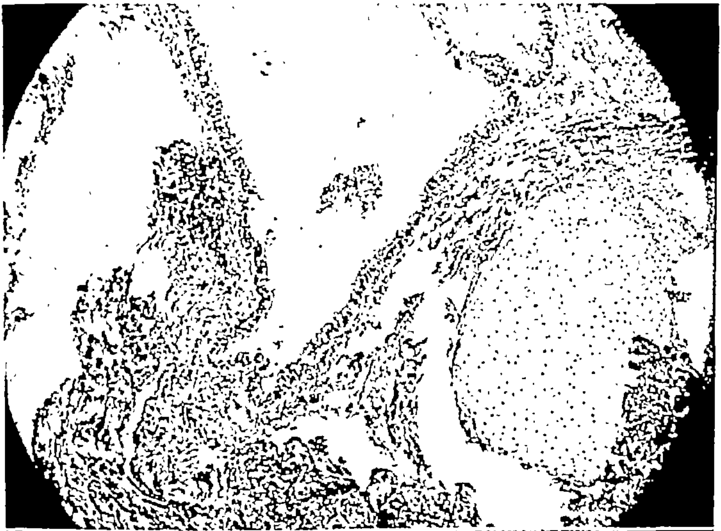


Fig. 8.—Illustrating bronchus and generalized emphysema of upper lobe (X79).

COMMENT

It has been assumed that the mesenchymatous anlage of a normal left lung gave rise to the two lobes of this specimen. The presence in the lower lobe of all the segmental bronchi normally found in an entire left lung has been interpreted as an ectopia bronchi of the left upper lobe. The posterosuperior segmental bronchi aerated but could not return air from the emphysematous, functionless upper lobe. Compression atelectasis of the lower lobe and mediastinal shift ensued. In the absence of a major bronchial communication from the upper lobe to the main bronchus it is impossible to explain the presence in that lobe of bronchi and of the bronchial cyst by the theory of lung formation propounded by Ekehorn[2] and Flint.[3] They described the development of branch bronchi as proceeding peripherally from the stem bronchus by a process of

Fig. 9.



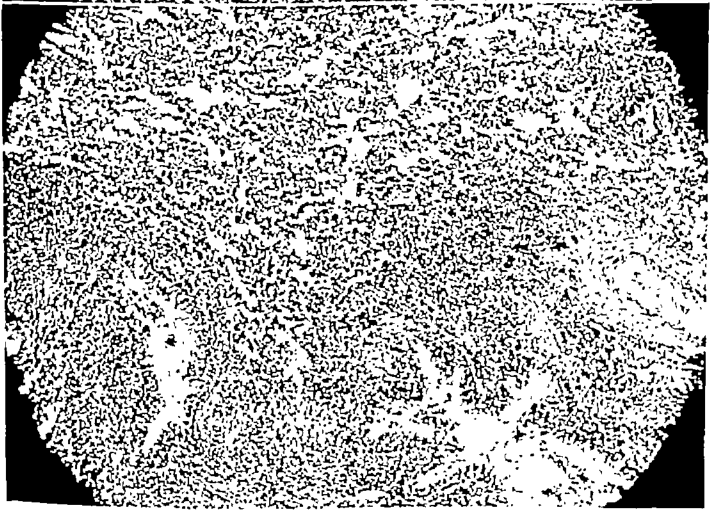


Fig. 10.

Fig. 9.—Illustrating bronchial epithelium and cartilaginous plate in wall of upper lobe cyst (×78).

Fig. 10.—Illustrating closely crowded bronchi and severe degree of lower lobe atelectasis (×78).

arborization. Heiss'[4] theory considers the stem bronchi as originating in the periphery of the lung and by a process of continuous infolding as giving rise to the connecting and terminal structures of the lung parenchyma. Such peripheral development has occurred in the case described; it but remained for the large bronchi arising centrally to join these smaller elements to complete a normal bronchial architecture of the upper lobe. Indeed, it would appear that here the mesenchymatous mass that Arey[1] denoted as contributing the nonepithelial elements to a lung had by a process of metaplasia given rise to bronchial epithelium. A more likely explanation is that the epithelium of the cyst and small bronchi arose from the bud of the left upper lobe bronchus, which bud later was incorporated into the lower lobe.

CONCLUSION

A case of ectopia bronchi of the left lung has been presented. Reference has been made to the prevailing theories of the development of the tracheobronchial tree and of the lungs, and the adequacy of these hypotheses has been questioned, particularly when reviewed in an effort to explain the anomaly described. If the knowledge of pulmonary embryology and the ability to diagnose lower respiratory tract anomalies is to keep abreast of recent advances in anatomy and surgery, detailed consideration must be given to a collection of many case reports such as that herein presented.

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LUNG HERNIA

A Case Report and Review of Literature

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THE occurrence of a case of lung hernia on the surgical service of this hospital has stimulated me to review the available literature. Lung hernia is a relatively rare condition. Less than 200 cases of lung hernia have been reported in the literature. A number of the earlier cases cannot be considered as true hernias of the lung but were merely traumatic eviscerations or prolapse.

A true lung hernia is defined as a protrusion of the lung and its pleural membranes through an abnormal opening in the thoracic enclosure.

The first case of lung hernia was reported by Roland in 1499.[9] However, this probably was not a true hernia of the lung. Montgomery and Lutz[6] credited Plater with the first report of a case of true lung hernia in 1641. Since that time there have been scattered reports of individual cases in the literature. Up to 1800 only fifteen cases had been reported, nine of which were true lung hernias. Morel-Lavallée[7] in 1849 compiled a detailed study of the cases, described the symptoms, and classified pulmonary hernias.

In 1925, Montgomery and Lutz in an extensive review of the literature found 165 cases. Goodman[3] in 1933 compiled 171 cases from the literature. Maurer and Blades[5] made the most recent study of lung hernia and reported seven additional cases—the largest individual report in the recent literature.

CASE REPORT

Mr. L. C. B., aged 46 years, a steamfitter, entered this hospital on March 28, 1946, with a history of having been in an automobile accident at 6 A.M. March 27, 1946. On admission the patient was found to have multiple lacerations and abrasions about the face, head, and left side of the chest; marked subcutaneous emphysema of the chest wall and supraclavicular areas; and multiple fractures of the left sixth, seventh, eighth, and ninth ribs posteriorly. There was considerable displacement of the fractured segments of the sixth and seventh ribs. A left hemothorax and hemopericardium ensued but under conservative therapy spontaneous recovery occurred except for slight pain and tenderness in the region of the fractured ribs.

On Jan. 16, 1947, the patient was readmitted to this hospital complaining of pain in the left posterior portion of the chest. The patient stated that following previous hospitalization he was unable to work because of pain. The pains were described as beginning beneath the left scapula and *radiating laterally to the left shoulder. The pains were burning in character,* constant, and aggravated by coughing. The patient also complained of dyspnea and slight hemoptysis on several occasions.

Physical examination on admission revealed a well-developed and well-nourished white man, ambulatory and not acutely ill. Temperature was 98.6° F., pulse 100, and respiration 20. The right pupil was contracted and the left pupil dilated; pupillary reflexes were absent. There was marked dental caries. The chest was barrel-shaped; a depression was noted beneath the inferior angle of the left scapula over the sixth and seventh ribs posteriorly. The depression was partially covered by the scapula. There was a palpable defect in the chest

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intratracheal anesthesia an incision was made over the seventh rib extending from the transverse process posteriorly to the posterior axillary line. The incision was extended down through the muscle and fascia until the pleura was reached. In portions the pleura was found directly beneath the skin. The hernial sac consisted of diverticular outpouchings of the pleura extending out over the posterior surface of the ribs. The sac was dissected free. The sixth rib was found to be fractured in two places and the distal end of the middle fragment was embedded in the pulmonary tissue underlying the fifth rib. This was freed and brought up into the operative field. The seventh rib was similarly fractured with the fragment displaced down under the eighth rib. This was likewise freed and brought up into the operative field. The eighth rib had been fractured but had healed with only a moderate amount of deformity. Adhesions of the lung to the hernial sac were found and freed and the lung allowed to retract into the pleural cavity. The hernial sac was then removed and the edges of the pleura were approximated with No. 000 chromic catgut sutures. The ends of the seventh rib were freshened. Holes were bored into each end and a fine stainless steel wire was used to approximate them. The sixth rib was similarly treated. This brought the ribs fairly well into a normal alignment. The overlying muscles and fascia were then closed with interrupted chromic catgut sutures. The skin was closed with interrupted black silk sutures. The patient received 500 c.c. of citrated blood and 1000 c.c. of glucose in saline during the operative procedure.

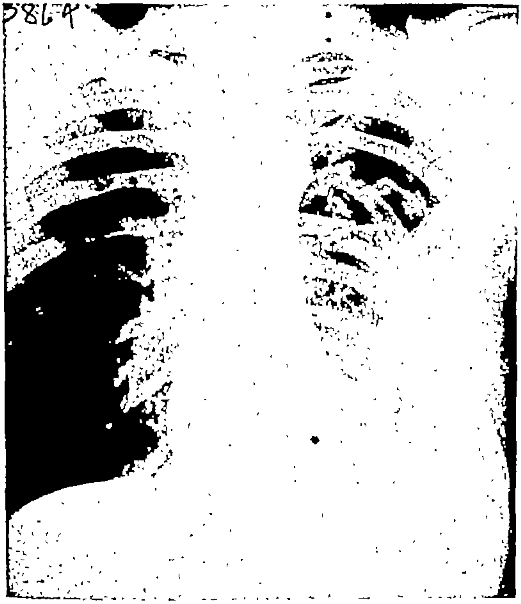

Fig. 2.—Postoperative chest x-ray view taken May 20, 1947. The defect in the thoracic cage has been corrected. Periosteal new bone formation is noted. There is mal-alignment of the fractures resulting from insufficient immobilization. Narrowing of the interspaces and thickening of the pleura in the left midportion and base are observed.

Postoperative course.—Shortly after the patient's return to his room respiratory embarrassment was noted, necessitating water-trap suction to reduce excessive pneumothoax;

suction was discontinued after two hours. General supportive therapy was instituted and penicillin was administered intramuscularly every three hours until Feb. 22, 1947. A moderately severe persistent cough associated with moderate mucopurulent sputum and left chest pains were noted postoperatively. X-ray examination of the chest on Feb. 4 showed some fluid in the left chest. This increased in amount, necessitating the aspiration of 870 c.c. of serosanguineous fluid on Feb. 10, 350 c.c. on Feb. 13, and 50 c.c. on Feb. 26; 100,000 units of penicillin were instilled into the pleural cavity after each thoracentesis. The sutures were removed on Feb. 10 at which time the wound edges appeared slightly reddened and indurated. The patient was allowed up in a wheel chair on the same date and soon thereafter was ambulatory. He developed a laryngitis on Feb. 20 which was treated with penicillin inhalations and responded slowly. The wound was well healed and when last seen in June, 1947, there was no evidence of recurrence of the lung hernia. The patient has vague chest and shoulder pains which seem to be greatly exaggerated. Re-check x-ray views on May 20, 1947, reveal some residual thickening of the pleura with narrowing of the interspaces on the left; at the site of the hernia repair there is some displacement of the rib fragments and evidence of periosteal new bone formation (Fig. 2).

DISCUSSION

The classification of lung hernias as devised by Morel-Lavalleé in 1849[9] and accepted by most authors is as follows:

- I. According to location:
  - A. Diaphragmatic
  - B. Thoracic
  - C. Cervical
- II. According to etiology:
  - A. Congenital
  - B. Acquired
    - 1. Traumatic
    - 2. Consecutive
    - 3. Spontaneous
    - 4. Pathologic

Korol,[6] Hartung,[5] and others question the occurrence of diaphragmatic lung hernia. There has been only one case reported in the literature. Beale[1] in 1882 reported the case of a 22-year-old white man who was pinned to the ground by a cart wheel striking him in the right abdomen. There was no external wound but autopsy revealed a defect in the right diaphragm through which the lung had apparently herniated with strangulation and abscess formation. Korol[6] considered this case as one of prolapse rather than herniation. Because of the pressure relationships, herniation of the lung through the diaphragm appears unlikely.

Korol[6] included mediastinal hernia in the category of lung hernia. A mediastinal lung hernia is a herniation of the lung through the mediastinal septum into the opposite hemithorax. Korol stated that it is the most frequent form of lung hernia, although few cases are noted in the literature to confirm this. It is said to occur frequently following modern collapse therapy (Korol, Hartung). The herniation occurs either anteriorly in the space between the sternum and the heart at the site of the atrophied thymus gland, or posteriorly between the esophagus and aorta below the level of the trachea and arch of the azygos vein. Mediastinal hernias are brought on by emphysema of one lung associated with contraction, atelectasis, or absence of the opposite lung. It may follow the abandonment of a long-continued pneumothorax. The pleural hernia of a tension pneumothorax is not to be confused with a mediastinal lung hernia.

Cervical lung hernias occur through the superior aperture of the thorax between the *sternocleidomastoid and scalenus anticus muscles and are due to a* tear or a deficiency of Sibson's fascia which normally limits the cervical excursion of the apex of the lung. Up to 1938 there had been only twenty cases reported in the literature.[6] They are usually said to be congenital.

The most common form of lung hernia is the thoracic or intercostal. Out of seventy-eight case reports reviewed by Montgomery and Lutz[9, 10] there were sixteen cervical hernias and sixty-two thoracic hernias. Thoracic hernias usually occur anteriorly near the sternum and posteriorly near the vertebrae. This is said to be due in each case to a locus minoris resistentiae in these areas, anatomically due to the fact that the external intercostal muscles extend forward only as far as the costochondral junctions and the internal intercostal muscles extend posteriorly only to the angle of the rib. Thoracic hernias are said to be more frequent anteriorly due to the fact that the pectoralis muscles cannot give as good support as the trapezius, latissmus dorsi, and rhomboideus muscles.

Montgomery and Lutz, in their review of 165 cases found in the literature, reported an incidence of 29 (18 per cent) congenital or early acquired hernias, 83 (50 per cent) traumatic, and 53 (32 per cent) spontaneous hernias. There has been some difficulty in determining whether a hernia is congenital or not. This is especially true when the hernia appears many months after birth. Hochsinger[11] stated that all hernias occurring in the first few weeks of life must be considered as congenital. Congenital lung hernias are due to several factors: (1) Weakness of the anatomic structures about the apex of the lung; (2) arrested growth of structures, that is, chest wall, sternal clefts; (3) intrauterine abnormalities such as pressure of an elbow against the fetal chest, amniotic bands, and pressure from tumors.

The great majority of acquired hernias are traumatic. A traumatic lung hernia may develop at any time after injury. Those first appearing late after injury have been classified by Morel-Lavalleé as consecutive hernias. Most writers agree that this subdivision is superfluous and merely group all such cases as traumatic hernias. Traumatic hernias may develop following operative wound, penetrating or bullet wound, or crushing injuries. Lung hernias are only infrequently noted following chest surgery. Careful subperiosteal resection of the ribs in thoracoplasty and other chest surgery probably accounts for the rarity of this complication.

Spontaneous lung hernias are the result of two factors, namely, a locus minoris resistentiae (that is, an area of local decreased resistance in the boundaries of the chest cavity) and an abnormal increase in the air pressure in the alveoli of the lungs. The abnormal increase in the intrathoracic pressure may be due to various factors. Paroxysmal coughing, which may be due to a variety of conditions such as chronic bronchitis, bronchiectasis, whooping cough, pulmonary abscess, and foreign bodies of the lung, is an important etiologic factor.

Occupations, such as glass blowing and playing of wind instruments, have been found etiologically responsible for a number of cases. Violent muscular exertion, such as heavy weight lifting or childbirth, is a third factor in the occurrence of lung hernias.

Pathologic lung hernias result from a variety of diseases of the chest wall which produce a locus minoris resistentiae. Abscess of the chest wall and breast, perforating lung abscess or empyema necessitatis, and malignancies have all been responsible for the occurrence of pathologic lung hernias.

According to Montgomery and Lutz, lung hernias are three times more frequent in males. Other authors report an even greater incidence in males.

The symptoms of lung hernias are varied. Goodman[3] listed three cardinal symptoms as follows: bulge, cough, and pain at the site of the bulge. The onset is usually insidious accompanied by pain and cough. The cough is chronic, spasmodic, and may or may not be productive depending on the underlying pathology. Early, the traumatic hernias are usually obscured by the more severe signs and symptoms of injury. The hernia may rarely become incarcerated or strangulated producing inflammatory changes.

On physical examination there is noted a bulging mass which increases on forced inspiration. A defect in the chest wall is usually readily demonstrable. When the hernia is protuded, the soft spongy crepitant consistency of the lung may be noted. A scar may or may not be present depending on the etiology.

X-ray and fluoroscopy may be of assistance in demonstrating the defect of the chest wall and an area of increased translucency may be noted outside the normal pulmonary boundaries. In mediastinal hernias there may be an asymmetry of the chest and on x-ray examination a large hyper-aerated lung is seen to extend into the opposite side in one or two bulgings having definite convex outer borders. The upper bulge is anteriorly between the first and fourth ribs and the lower bulge is posteriorly between the fifth rib and diaphragm. The trachea and esophagus are displaced to the contracted side.

The diagnosis of lung hernia is not difficult if the condition is kept in mind. The physical findings are readily apparent. The differential diagnosis should be made from subcutaneous emphysema, perforating empyema, aneurysm of the intercostal vessels or aorta, fibroma, lipoma, and hemangioma. Mediastinal lung hernias must be differentiated from the pleural hernia of a tension pneumothorax in which there is no lung herniation present.

Complications are rare. Strangulation and gangrene of the lung may occur. Hemoptysis is not infrequent. The prognosis of lung hernia is good; it is rarely fatal, but rarely heals spontaneously.

The treatment of lung hernias is either medical or surgical. Medical treatment is in the main palliative consisting of rest, change of occupation, and local compression with pads and elastic devices. With palliative treatment the defect in the chest wall may become increasingly rigid, but the hernia, although smaller, will still persist; an increase in intrathoracic pressure will cause a return and increase in the size of the protusion. Surgery is contraindicated in a number of cases due to the underlying pulmonary pathology and general condition of the patient.

Surgery offers the only hope for a permanent cure. Various operative procedures have been devised. Vogler[5] in 1898 performed a plastic operation using periosteum or bone flap from the sternum. Vulpius[8] in 1898 performed a plastic operation in which he crossed strips of ribs which were sutured in place with silver wires; this hernia recurred in one year. Tuffler[4] in 1891 reported a cure by freeing and ligature of the hernial sac. Graham[4] in 1922 treated a spontaneous cervical hernia associated with an adenoma of the thyroid by excision of the adenomatous thyroid and packing of the wound with iodoform gauze. The packing was removed on the fifth postoperative day and the wound healed without recurrence of the hernia. Montgomery and Lutz[9] in 1925 reported a case of traumatic intercostal hernia cured by an osteoplastic procedure.

Goodman[3] in 1933 treated a traumatic consecutive intercostal hernia in a 42-year-old seaman by periosteal plastic closure. The redundant pleura was resected and closed with continuous catgut suture following which the periosteum of the rib above the defect was reflected downward, the periosteum of the rib below the defect was reflected upward, and both were sutured over the defect with chromic catgut.

Winkel[14] in 1935 reported a cure in a traumatic intercostal hernia by a repair consisting of weaving of fascia lata strips across the defect.

Repair of the defect with tantalum plate has been attempted by Paulson[8] but has been found unsatisfactory because of difficulty of immobilization of the chest wall.

These have in the main been individual case reports. Maurer and Blades[8] have undoubtedly had the largest individual experience with the surgical treatment of lung hernias in recent times. In 1946 they reported seven cases of traumatic intercostal lung hernias, four patients of whom submitted to surgery consisting of closure by periosteal flaps. In addition, they reported four cases of herniation of the liver and peritoneum through the lower thoracic cage which were repaired in a similar manner. Results have been excellent. Maurer and Blades believe that any defect of the chest wall regardless of size can usually be repaired by plastic procedures utilizing only structures which are a part of the chest wall, namely the rib, periosteum, and muscle. The most important feature is closure of the defect with sturdy bone or periosteum which will produce bone. The freeing of adhesions and of the lung about the margins of the defect with the creation of a local pneumothorax is important in the production of a cushion between the lung and the freshly repaired chest wall. This serves to reduce to a minimum the possibility of the lung being forced into the chest wall by coughing and straining during the immediate postoperative course.

SUMMARY

A review of the available literature concerning lung hernias has been presented.

A true lung hernia is a protusion of the lung and its pleural membranes through an abnormal opening in the thoracic enclosure. Its classification, etiology, symptoms, and treatment have been reviewed. The diagnosis is not difficult provided the condition is kept in mind.

The acute experiment was performed under sodium pentothal-sodium barbital anesthesia (sodium pentothal 20 mg. per kg., sodium barbital 150 mg. kg.). A rubber catheter was passed through the external jugular vein into the right ventricle under fluoroscopic guidance. At the time the dog was under the fluoroscope the state of the heart and lungs was noted. In no animal was there an accumulation of pleural fluid. Residual pneumothorax was minimal.

Right intraventricular pressure was measured through the intracardiac catheter by a water manometer; mean systemic blood pressure was measured from a cannula in a femoral artery. Cardiac output was estimated by the direct Fick principle, mixed venous blood being withdrawn from the catheter in the right ventricle and arterial blood being taken from the uncannulated femoral artery by needle puncture. Blood oxygen content was determined in duplicate by the method of Van Slyke and Neill. Oxygen consumption was measured continuously with a modified Roth-Benedict spirometer. An endotracheal catheter attached directly to the spirometer eliminated the necessity for tracheotomy. Leakage of air around the catheter was prevented by inflating a thin rubber cuff fitted to the outside of the catheter. This method made it possible to record any changes in tidal air and rate of respiration as well as oxygen consumption.

Records were made until the dog was in a steady state, usually one to two hours after the anesthesia had been given. Simultaneous arterial and right ventricular blood samples were then collected for estimation of the cardiac output. The ligature about the lung root was tightened by pulling on the end protruding through the chest wall. Occlusion was complete in 4 to 8 seconds. During this period continuous recordings were made of oxygen consumption, tidal volume, right intraventricular pressure, systemic blood pressure, and Lead II of the electrocardiogram. Within 5 minutes of ligation samples of blood were again drawn for the second cardiac output determination. The recordings were continued one to two hours or until the animal died.

A total of twenty dogs were studied. Autopsies were done in all cases. The condition of the lungs, the effectiveness of the ligature about the lung root, and the state of the heart were observed. In all instances the ligature effectively occluded the bronchus and accompanying blood vessels.

RESULTS

The sudden ligation of one lung root caused an immediate rise in right intraventricular pressure of from 20 to 129 mm. of water (average 57 mm.) lasting 3 to 60 minutes (average 25 minutes). There was a transient slowing of the heart for 4 to 5 beats but no other change in the electrocardiogram. Systemic blood pressure showed a transient fall of 10 to 15 mm. Hg during the time the ligature around the lung root was being tightened but rose again to the preligation level and remained steady (Fig. 1).

In eight dogs the changes in cardiac output after ligation of the lung root were no more than ± 200 c.c. per minute (from ± 5 to 17 per cent of the control value), and these were considered insignificant variations. (Significance was based upon both the limits of accuracy of the method for determination of oxygen content of the blood samples and changes in output which might reasonably be expected to occur over a ten-minute period under barbiturate anesthesia.[9, 10]) In three dogs there were increases in cardiac output of 400 c.c., 800 c.c., and 1,900 c.c. per minute (+ 25 per cent, 32 per cent, and 63 per cent). In six instances the output decreased 300 c.c. to 700 c.c. per minute (18 to 45 per cent), and in two the outputs fell 1,600 c.c. and 1,900 c.c. per minute, decreases of 53 per cent and 68 per cent.

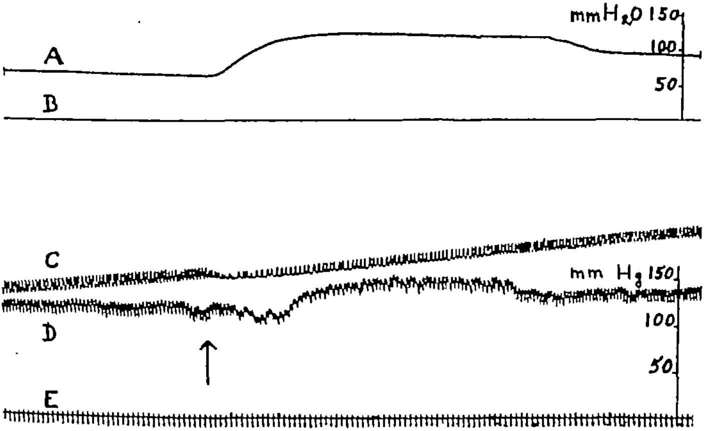

Fig. 1.—Effects of ligation of right lung root (Dog 16) at arrow. *A*, Right intraventricular pressure; *B*, base line at level of right atrium; *C*, respiration and oxygen use; *D*, mean arterial blood pressure (femoral artery); *E*, base line for blood pressure, 4 second intervals.

The oxygen consumption before and after ligation remained essentially the same. In twelve dogs the minute volume of respiration did not change significantly; in four of these neither the tidal air nor the rate of respiration changed, and in eight an increase in rate of respiration was proportional to a decrease in tidal air. (Significance was based on the fact that, with the spirometer used, tidal air could be estimated within 10 c.c. A change in minute volume of less than ten times the rate of respiration was considered insignificant.) The minute volume decreased 300 to 800 c.c. (19 to 25 per cent) in four dogs; in three of these this was due to a decrease in tidal air without a completely compensatory increase in rate, and in one exceptional case in which respiration became suddenly slower and deeper the decrease in rate (41 per cent) was greater than the increase in tidal air (37 per cent). The minute volume increased 1,000 to 1,100 c.c. (30 to 44 per cent) after ligation in four instances due to a very large increase in rate of respiration (25 to 59 per cent) out of proportion to any change in tidal air.

In the twelve dogs with no significant change in over-all minute volume there was no significant change in the efficiency of respiration as measured by the ventilation equivalent. (Ventilation equivalent = liters of air necessary to be ventilated for the absorption of 100 c.c. of oxygen.[8]) With an increase in minute volume, the efficiency decreased (ventilation equivalent increased); with a decrease in minute volume efficiency of oxygen absorption increased (ventilation equivalent decreased).

TABLE I. CARDIOVASCULAR FUNCTION BEFORE (A) AND AFTER (B) PNEUMONECTOMY

		CARDIAC OUTPUT (LITERS PER MIN.)			STROKE VOL. (C.C.)		PULSE		RT. VENT. PRESS. MM. OF H_2O			
NO.	LUNG	A	B	PER CENT CHANGE	A	B	A	B	RISE	TIME TO RETURN (MIN.)	ART. B.P. CHANGE	AUTOPSY ABNORMAL FINDINGS
1	RPA	1.9	2.0	+ 5								
2	LL	1.2	1.0	-17								
3	RL	1.9	1.8	- 5								RLL abscess
4		2.5	3.3	+32								
5	RL	0.7	0.4	-42	5.4	2.7	140	148				
6	RL	1.1	0.6	-45	6.5	3.2	168	190				
7	RL								28	8		
8	RL	2.1	1.9	- 9	17.5	16.5	120	114	81	60		LUL, LML atelectatic
9	LL	3.0	4.9	+63	18.7	36.2	160	135	36	15		LLL atelectatic
10	LL	2.2	2.1	- 5	14.5	14.0	160	150	None			
11	RL	0.5	0.3	-40	3.8	2.1	130	140	70	30	No change	RLL, atelectatic
12	RL	1.2	1.2	0	6.3	7.6	190	170	80	30		
13	RL	2.8	0.9	-68	16.5	5.3	170	170	105	30	Fell, Died	Infarct RLL, RML, worms right heart
[illegible]	RL	1.8	1.3	-27	9.5	8.1	190	160	20	15		Infarct RLL
[illegible]5	RL	2.1	1.9	- 9	10.0	7.6	210	250	55	30	Sl. fall	
16	RL	3.8	3.1	-18	23.4	17.2	162	180	117	25	No change	Infarct RLL
17	RL	3.0	1.4	-53	20.0	10.0	150	140	59	60	Fell	
18	RL	1.5	1.9	+26	10.0	12.6	150	150	40	17	No change	
19	RL	2.2	1.7	-23	15.7	14.2	140	120	40	7	Sl. rise	Catheter Rt. atrium
20	RL	1.2	1.3	+ 8	7.6	8.7	170	150	20	3	No change	

At the end of the acute experiment eighteen of the twenty dogs had apparently compensated for the functional removal of one lung. The blood pressure had been maintained and there was no respiratory difficulty. Two failed to compensate. One of these showed a decrease in cardiac output from 2.8 to 0.9 liters per minute, the stroke volume falling from 16.5 to 5.3 c.c. Systemic blood pressure fell immediately from 72 mm. Hg to 54 mm. Hg, rose to 86 mm. and then fell gradually until death occurred at the end of one hour. At autopsy the heart was found to be dilated and the right heart and pulmonary artery filled with the round worms *Dirofilaria immitis.* On section the blood vessels in the lung were also found to be full of microfilariae of *D. immitis.* The cardiac output of the second dog fell from 3.0 to 1.4 liters per minute, the systemic blood pressure decreased gradually from 130 to 64 mm. Hg, and Traube-Hering waves appeared. After two hours the animal appeared unlikely to recover and was sacrificed. At autopsy the heart and lungs were grossly normal.

DISCUSSION

The conditions under which the experiment was carried out were admittedly not entirely normal. Barbiturate anesthesia is known to decrease the cardiac

TABLE II. PULMONARY FUNCTION BEFORE AND AFTER PNEUMONECTOMY

NO.	LUNG	O_2 USE C.C. PER MIN.		MIN. VOL. LITERS PER MIN.		TIDAL AIR (C.C.)		RESP. RATE PER MIN.		VENT. EQUIV. L. PER 100 C.C.		AUTOPSY, ABNORMAL FINDINGS
		BEFORE	AFTER	BEFORE	AFTER	BEFORE	AFTER	BEFORE	AFTER	BEFORE	AFTER	
1	RPA	62	62	4.5	4.5	42	42	108	108	7.3	7.3	
2	LL	35	35	3.7	4.7	83	52	45	88	1.1	1.3	
3	RL	42	44	2.7	2.7	71	71	38	38	6.4	6.1	RLL Abscess
4		56	62	2.5	3.6	67	73	38	49	4.5	5.8	
5	RL	52	53	3.6	3.2	83	54	43	59	6.9	6.0	
6	RL	30	30	1.6	1.3	72	52	22	25	5.3	4.3	
7	RL	46	43	2.0	2.2	100	100	20	22	4.4	5.1	
8	RL	60	67	3.4	3.0	62	52	55	57	5.7	4.4	LUL, LML atelectatic
9	LL	74	74	3.7	3.0	83	114	44	26	4.9	4.0	LLL atelectatic
10	LL	53	53	2.8	3.8	104	98	27	39	5.3	7.2	
11	RL	25	25	3.6	4.7	108	93	33	50	1.4	1.9	RLL atelectatic
12	RL	52	59	2.7	3.8	93	83	29	46	5.2	6.5	
13	RL	64	57	3.4	3.0	89	72	38	42	5.3	5.3	Infarct RLL, RML, worms right heart
14	RL	49	46	1.9	1.3	62	39	30	32	3.8	2.7	Infarct RLL
15	RL	242	242	3.7	4.3	62	55	60	78	1.5	1.8	
16	RL	83	85	3.0	3.2	166	145	18	22	3.6	3.8	Infarct ½ RLL
17	RL	62	55	1.3	0.7	35	13	36	51	2.0	1.2	
18	RL	83	83	3.2	2.4	83	52	39	46	3.9	2.9	
19	RL	83	71	2.2	2.0	42	42	53	48	2.7	2.8	
20	RL	73	80	3.6	3.7	125	93	29	40	5.0	4.7	

output of an animal.[9, 10] According to Shore and associates[9] the decrease in cardiac output with anesthesia is neither constant nor predictable. Over 20 to 30 minute periods they cite decreases of from 10 to 30 per cent, and, on the other hand, over some 90 minute periods they have found decreases of only 6 to 7 per cent. In our studies the two estimations of cardiac output were made not more than 10 minutes apart, and at least 60 minutes after induction, hence, since the animal was in a steady state with regard to blood pressure and oxygen consumption when the ligature was tightened, it may be assumed that any change in cardiac output more than 10 per cent in the period immediately following could be attributed to the manipulation.

The experiments were done with the chest closed to enable a continuous recording of both oxygen consumption and the details of pulmonary ventilation. The dog was breathing against a definite resistance at all times, but this resistance was constant, and relative changes in the character of respiration were readily seen.

Since we found the right pulmonary artery to be slightly larger in diameter than the left, the right side was used after the first few experiments as the side which might be expected to give the greater changes.

Compensatory circulatory and respiratory mechanisms were sufficient in all but two dogs to maintain the systemic blood pressure and the oxygen consumption virtually unchanged. This speaks for the large reserves of the vascular bed and airways of one lung which can immediately take over the functions of two.

The outstanding change in the cardiovascular system, which occurred in all but one instance, was an increase in right intraventricular pressure. This rise was relatively slow in development, taking from 2 to 10 minutes to reach its peak, remaining on a plateau for 10 to 15 minutes, and gradually falling to the preligation level in 25 to 30 minutes. It was not correlated with changes in cardiac output, stroke volume, or systemic blood pressure. One might expect that the sudden removal of a large part of the pulmonary capillary bed would result in greatly increased resistance in the pulmonary circuit. The return of the ventricular pressure to its former level suggests that additional vascular channels were opened, or that existing vessels increased in size under the increased pressure. This opening of a reserve vascular bed has been postulated previously by Tigerstedt[11] and by Wiggers.[12]

The cardiac output following compression of the pulmonary artery has been of interest to many observers. In 1907 Tigerstedt,[11] measuring the minute output of the left heart with a stromuhr, found that it was only slightly decreased by ligation of the left pulmonary artery. Haggart and Walker,[13] in nine experiments on cats, also found only a slight decrease in cardiac output with compression of the left pulmonary artery. Fineberg and Wiggers[6] found decreases in left ventricular output with compressions of the pulmonary artery exceeding 41 to 72 per cent (average 58 per cent).

In the present studies there was no change in cardiac output in eight experiments. In three the output increased. In eight there was a significant decrease in cardiac output following ligation of a lung root. With a decrease in cardiac output up to 45 per cent, compensatory mechanisms were adequate to prevent a change in systemic blood pressure. In the two cases in which the cardiac output fell 53 and 68 per cent the arterial blood pressure fell steadily over the remainder of the experimental period.

The changes in cardiac output, when they occurred, appeared to depend largely on changes in stroke volume. Although the heart rate was usually somewhat altered by ligation of the lung root (average change plus or minus 19 beats per minute), the direction of change was unpredictable and the magnitude not sufficient in itself to effect a significant increase or decrease in cardiac output if not associated with a similar change in stroke volume. Out of a total of fifteen dogs, eight showed changes in stroke and pulse opposite in direction; five resulted in significant changes in output in the direction of the change in stroke volume. Two dogs maintained a constant heart rate but variations in stroke volume were sufficient to bring about significant alterations in cardiac output. In three instances in which there was a significant decrease in cardiac output, changes in stroke volume and heart rate were parallel.

With ligation of one lung root a certain volume of blood is trapped in the lung. What part this plays in the reductions of cardiac output in these experiments is not known.

There was a transient drop in systemic blood pressure which occurred as the ligature was being pulled tight. Parin[14] has described a fall in systemic blood pressure which he believes due to a reflex mediated through the vagus nerve from pressoreceptors in the pulmonary artery. The pressures used by Parin experimentally were 40 mm. of Hg or above. In the present studies the pressures in the right ventricle, and therefore in the pulmonary artery, did not approach 40 mm. of Hg. Furthermore, the transient fall in blood pressure did not coincide with the rise in right intraventricular pressure but preceded it. The transient blood pressure drop did coincide with a brief slowing of the heart, and was probably caused by vagal stimulation set up by tugging on the lung root. Vagal arrest never occurred.

The adjustment of the respiratory system to the sudden removal of one functioning lung was complete in that oxygen continued to be absorbed at a constant rate. It is obvious, then, that the remaining lung began immediately to absorb as much as double the oxygen it had absorbed prior to ligation.

The changes in ventilation of the remaining lung were unpredictable. Theoretically, oxygen consumption could be increased by either increasing the minute volume of respiration or the efficiency of oxygen absorption or both.

In over one-half the dogs the recorded minute volume did not change, which means, in actual fact, that in these cases the minute volume of the one remaining lung approximately doubled. Two factors determine minute volume: rate of respiration and tidal air. Over-all minute volume was maintained unchanged either by a large increase in tidal air of the remaining lung or by a smaller increase in tidal air together with an increase in respiratory rate.

In other dogs there was altered efficiency of oxygen absorption, as m by the ventilation equivalent, due to changes in the minute volume af tion and no real change in oxygen use. Since the ventilation equivalent is a ratio between the minute volume and the oxygen uptake, it varies as these two factors vary. The rate and depth of respiration are controlled by complex nervous and chemical mechanisms. Oxygen absorption in the lungs, on the other hand, is a physical process depending upon such conditions as the permeability of the alveolar and capillary membranes, the area of the absorbing surfaces, the time of exposure of the blood to the alveolar air, the total volume of blood exposed to the alveolar air in any period of time, and the difference in partial pressure of the oxygen of the alveolar air and the blood of the pulmonary artery. Minute volume and oxygen consumption may vary independently, and the ventilation equivalent may be affected by alterations in either. For example, in Dog 18 the ventilation equivalent before ligation was 3.9, for 30 seconds after ligation the tidal air was very small and the rate of respiration slow, but oxygen use was not reduced proportionally to minute volume. The ventilation equivalent at this time was 1.8. Respiration gradually became deeper and faster; oxygen consumption returned to the preligation level and the ventilation equivalent became 2.9. These changes in ventilation equivalent denoting altered efficiency of oxygen absorption per liter of ventilated air depend upon reduction of minute volume of respiration rather than changes in oxygen uptake following ligation. Oxygen absorption was but little affected by the operative manipulation, whereas respiratory movements were momentarily inhibited, presumably through nervous reflex mechanisms.

Since the ventilation equivalent which relates the two factors of minute volume and oxygen uptake is not a constant, it does not appear to be a necessarily reliable index of respiratory function, at least when conditions are basal. There is, of course, a limit to the capacity of a lung to absorb oxygen. This limit may be reached more quickly in lungs with diseased alveolar membranes or greatly decreased alveolar surfaces. To approach such a limit would mean increasing the oxygen demand until the lowest possible ventilation equivalent had been attained. Under such conditions of stress the ventilation equivalent might prove to be a better criterion of cardiorespiratory function.

These studies do not indicate to how much additional stress, such as exercise or an increased blood volume, an animal could adjust after pneumonectomy. Longacre and associates[15, 16] found some indication of the loss of cardiorespiratory reserve after pneumonectomy in chronic experiments measuring responses of dogs to exercise, using pulse, respiratory rate, body temperature rise, and arterial oxygen saturation as criteria of function.

Ligation of one lung root does put increased stress on the right ventricle. Right intraventricular pressure increased in all but one of the cases measured. Fineberg and Wiggers[6] have found that the right ventricle, like the left, is able to meet increased peripheral resistance by an increased force of contraction, but that the range of compensation is extremely narrow. Almost all of our dogs were able to adjust to the added stress put on the right ventricle. Although 40 per cent showed an immediate reduction in cardiac output, systemic blood pressure was maintained. What happened to the cardiac output in the succeeding hours was not determined. Tying off the right branch of the pulmonary artery is equivalent to occlusion of at least 50 per cent of the pulmonary artery. Fineberg and Wiggers found one dog in their series which was unable to maintain right ventricular output unchanged when 41 per cent of the pulmonary artery was occluded. One dog in our series with apparently normal heart and lungs was unable to maintain the systemic blood pressure after sudden pneumonectomy.

With the pulmonary vascular bed cut in half, it might be expected that the pressure in the pulmonary circuit would be increased more readily. Gibbon, Gibbon and Kraul[17] showed that in cats when the right middle and lower lobes and all of the left lower lobes were removed a blood transfusion of 15 c.c. per kg. caused death due to pulmonary edema in four out of nine animals. A similar type of study before and after pneumonectomy might prove enlightening.

In conclusion, the majority of dogs in this series were able to make physiologic adjustments to the functional loss of one lung; two dogs, one with heart disease and one with apparently normal heart and lungs, were not. No attempts were made to measure the cardiorespiratory reserves after such a procedure.

SUMMARY

1. The details of cardiovascular and respiratory adjustment to functional pneumonectomy were studied in twenty dogs with closed chests. Pneumonectory was accomplished under barbiturate anesthesia by the sudden ligation of one entire lung root.

2. The immediate response of the cardiovascular system was a rise in right intraventricular pressure, and, in 40 per cent of the dogs, a decrease in cardiac output. Systemic blood pressure was usually maintained.

3. Oxygen consumption before and after ligation was essentially unchanged.

4. The changes in pulmonary ventilation were unpredictable and consisted of increases in tidal air of the remaining lung, increases in rate of respiration, and altered efficiency of oxygen absorption.

5. Two dogs were unable to compensate for this functional pneumonectomy. One showed cardiac disease, and one had apparently normal heart and lungs.

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THE SEGMENTAL AND LOBULAR PHYSIOLOGY AND PATHOLOGY OF THE LUNG

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THE introduction of the concept of segmental resection of the lung opened what was referred to at that time as an unwritten chapter in surgical anatomy. This chapter is now quite complete, and gratitude is expressed to Brock, Blades, Huber, Boyden, Scannell, and others for their painstaking contributions to descriptive anatomy. Mastery of the three dimensional intricacies of the interlacing bronchi and blood vessels is now an essential step toward the attainment of the precision required in present-day pulmonary surgery Attention is now directed to another chapter that awaits development—the segmental and lobular physiology and pathology of the lung. Some of the material is available and a firm structural basis has been provided by William Snow Miller, but the parts remain insufficiently related and incomplete. While the concepts that are certain to emerge may not guide our dissections, they will provide us with methods that are based on a more complete understanding of the organ with which we work.

Tribute is paid immediately to the brilliant contribution of Van Allen and Lindskog on collateral ventilation and the aerodynamics of bronchial obstruction presented to this Association many years ago. I shall return to this later on, but wish to comment now on the fact that this discovery was made by surgeons. Physiologists have been slow to accept the results of the easily repeated experiments and the significance of the mechanism has never received the attention it deserves. Possibly one reason is that physiologists are in the habit of dealing with the two lungs as a single organ. It has been their custom to select the trachea as the site to introduce resistance to the entrance and exit of air when experiments are designed to study the aerodynamics of respiration. Similarly the analysis of pulmonary edema has been approached through the mimicry of cardiac failure with abnormal conditions imposed upon the pulmonary circulation

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as a whole, or through the inhalation of irritant gases that are dispersed throughout the entire respiratory tract.

The bronchopulmonary segment with its component lobules is a physiologic unit of the lung just as the segment is a surgical unit of the lung and has become a unit of roentgenologic diagnosis. At the level of the segment or the lobule, obstruction to the outflow of air produced by disease or by neuromuscular action can produce volume and tension changes within a limited lung area at the expense of volume alterations in adjacent areas. The potentialities of this mechanism are lost sight of when all lobules and segments are subjected to identical conditions, which is the case when obstruction or resistance is imposed at the level of the trachea. The discovery of collateral air drift by Van Allen and Lindskog brought emphasis to this important difference.

The increased tension of the air within alveoli that is produced by partial occlusion of the bronchus to an entire lobe is referred to clinically as obstructive emphysema. The mechanism of its production by partial occlusion of the lumen and a widening of the air passages on inspiration and a narrowing on expiration is understood. The basic condition is obstruction to the outflow of air. Similarly, the acute lobar collapse caused by complete bronchial obstruction and absorption of the alveolar air is recognized experimentally and clinically. Both the collapse and the obstructive emphysema that might be expected to occur by the same mechanisms acting within the bronchopulmonary segment are subject to the special conditions imposed by collateral air drift and also by the secretory activity of the bronchial mucous membrane. These will be discussed. I shall, however, begin with the single lobule or a cluster of adjacent lobules and consider the significance of increased tension of the air within the alveoli and a possible relationship to the onset of pulmonary edema.

The several factors that control the passage of exudates and transudates across the wall of the capillary need not be enumerated. They are itemized in a diagram by Drinker (Fig. 1). Attention will be confined to the physical force studied by Landis and Gibbon[1] that assists in the control of filtration edema in the extremities, increasing tissue pressure. These authors measured the effects of the pressure that is built up by an increasing volume of fluid in the tissue spaces as the elasticity of separated tissue elements is encountered. This pressure opposes the filtration pressure within the capillary and slowly brings the leakage from capillaries to a halt. When one attempts to visualize the possibility of such a force acting in pulmonary tissue it is apparent that the unique structure of the lung creates a set of conditions different from those that exist in the tissues of the extremities. The lung tissue may undergo alterations in volume without creating pressure changes of any considerable magnitude. Tissue pressure built up by fluid escaping from the capillaries is a control measure that could be effective in the lung only when carried to the extreme of lobar consolidation, at which point the effect of the limiting membrane of the visceral pleura might be felt. In any event, the control force studied by Landis and Gibbon would be intolerable in the respiratory organs, for leakage from pulmonary capillaries into alveolar spaces is immediately destructive to the primary function of the lung.

In the lung the equivalent of mounting tissue pressure from fluid may be found in increased tension of the alveolar air. As long as the increased tension is confined to a lobule or segment, this portion of lung can distend through encroachment on adjacent lung without appreciable respiratory embarrassment. Were the mechanism activated in all portions of the lung simultaneously, a crisis of labored breathing, anoxia, and, at times, death would be expected, as is the actual fact in bronchial asthma.

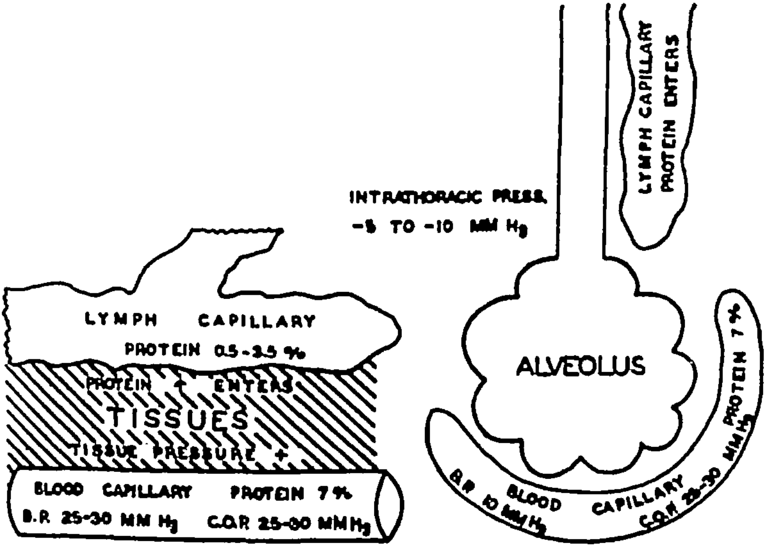


Fig. 1.—Diagrams of contrasting fundamental physiologic relations between blood capillaries and tissues. On the left, a representation of typical relations in the skin; on the right, relations in the lungs. (From Drinker: Pulmonary Edema and Inflammation, Harvard University Press.)

To postulate this happening in a portion of lung threatened by edema requires an explanation for the occurrence of obstruction to the outflow of air from a single area. This is known to occur locally in tension cysts and cavities, but in such instances is usually traced to a valve-like fold of mucous membrane or other "ball-valve" mechanism inherent in the pathologic lesion. In normal lung this action could arise from local reflex activation of the neuromuscular apparatus of the bronchi, for the vegetative neuromuscular apparatus of the human lung appears concentrated in the bronchial tree. It is true that the pulmonary arteries are possessed of smooth muscle but the weight of present evidence indicates that the lesser circulation is regulated in a passive manner except perhaps under certain emergency situations. The elaborate neuromuscular structure that surrounds the air passages has been described in detail (Fig. 2). The constant and vigorous activity of this apparatus has been expressed by Macklin[2] in the concept of the dynamic bronchus. Far from being rigid tubes, the bronchi undergo inspiratory dilatation with lengthening and expiratory construction with shortening. The walls of the alveoli do little or no stretching and the factor of safety of their elastic tissue is said to be slight.

The bronchial musculature is projected along the bronchiole and respiratory bronchiole to form sphincters about the openings into the atria. Sphincters are also formed about the openings into the alveoli that arise directly from the respiratory bronchiole. Trapping of air in the alveoli by obstruction to its outflow may well result from the sphincteric action of the bronchiolar musculature. The increased tension of the trapped air is dependent upon cough or expiratory grunting. Cough is known to have a twofold effect: The forcing of air into alveoli by the increased pressure built up within the air passages, as well as the expulsion of mucus from the upper portions of the tracheobronchial passages that comes on release of the laryngeal sphincter. Fluoroscopists are completely familiar with the increased radiance of the apical segments when they "light up" with cough; also with the dispersion of lipiodol from bronchi into alveoli

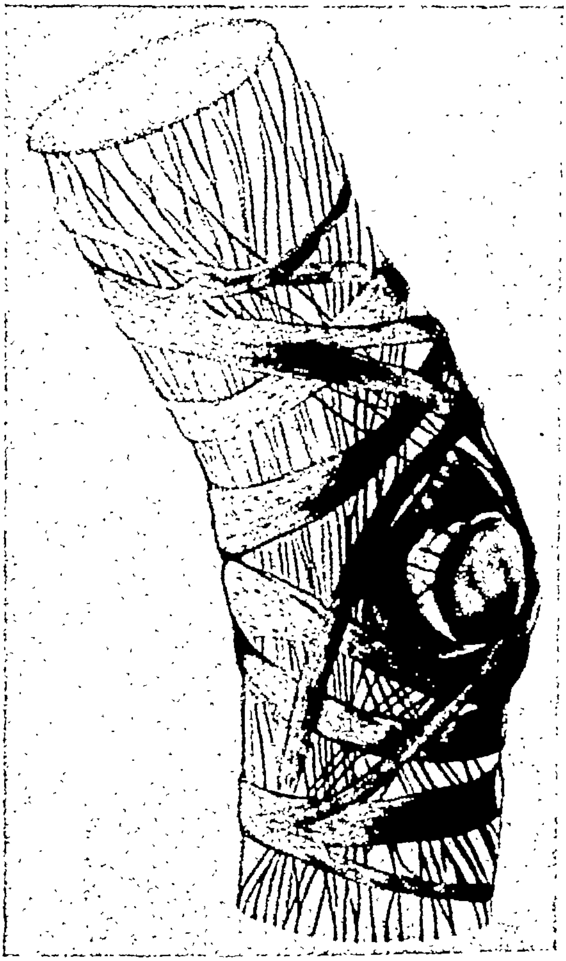


Fig. 2.—Arrangement of smooth muscle bands and elastic fibers. (From Miller: The Lung. Charles C Thomas, Publisher.)

with cough. The irritative nonproductive cough that attends the onset of pneumonia or ushers in a paroxysm of asthma thus provides a protective measure that builds up air tension in alveoli to support the capillary walls by external pressure.

The sequence of events may be pictured somewhat as follows (Fig. 3): (1) inspiratory widening of the terminal bronchioles; (2) early in expiration the quick closure of the laryngeal sphincter with elevation of endobronchial air tension by the muscular effort of a cough or grunt; (3) trapping of air in lobules under increased tension; (4) slow reduction in the tension by collateral air drift through the pores so that on the next cough the lobule receives a blast of fresh air with renewed oxygen supply.

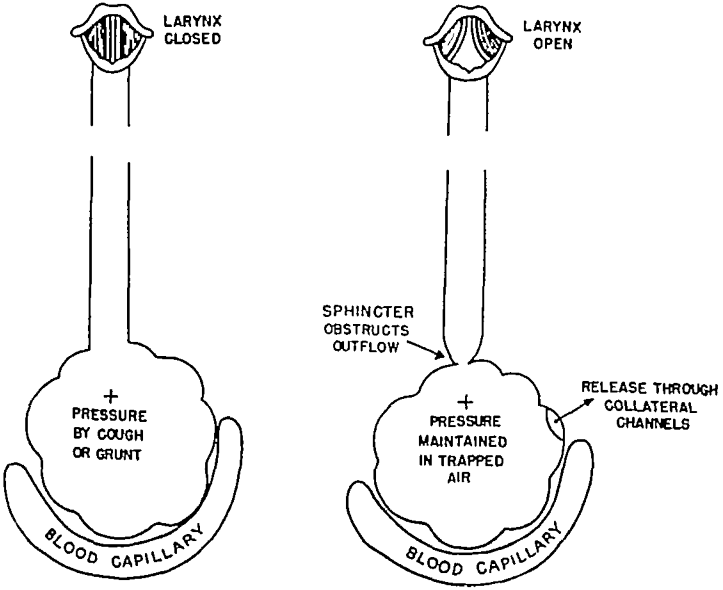

LARYNX CLOSED
+
PRESSURE BY COUGH OR GRUNT
BLOOD CAPILLARY
LARYNX OPEN
SPHINCTER OBSTRUCTS OUTFLOW
+
PRESSURE MAINTAINED IN TRAPPED AIR
RELEASE THROUGH COLLATERAL CHANNELS
BLOOD CAPILLARY

Fig. 3.—Diagram representing mechanism to establish and maintain intra-alveolar air pressure supporting capillary wall in a segment or subsegment of lung.

Surgeons of World War II, faced by localized pulmonary edema resulting from the direct trauma of a chest wound, found that the effective course of management lay in clearing the upper respiratory passages, controlling pain by intercostal nerve block, and aspirating pleural blood and air that might be limiting lung expansion. The cough reflex was not suppressed by morphia nor was the chest wall motion restricted by tight strapping. By these measures the wet massive collapse of the lung seen in World War I was practically unknown.

The use of a gaseous medium under tension as an extrinsic means of applying increased pressure to support the capillary wall provides the theoretical basis for administering oxygen under pressure in the treatment of irritant gas poisoning and other forms of generalized pulmonary edema as recommended by Barach. The intrinsic mechanism was forecast by Keith in 1909[3] and also by Miller,[4] who stated that, "the action of the bronchial musculature in expiration is not passive, but active, and its action in regulating the tensions within air sacs deserves more attention than physiologists have given to it." Surgeons who work daily with the human lung are well aware of the seeming vagaries of lobular and segmental inflation and deflation. Local reflex changes in circulation that are commonly observed in the abdominal viscera as a result of manipulation and exposure are not discernible in the lung. It is possible that the commonplace local changes in ventilation may have a similar origin and significance. Again, the experienced thoracic surgeon holds great concern for the immediate restoration of the cough reflex after it has been temporarily suspended by anesthesia. This is usually rationalized on the basis of clearing the major air passages. Perhaps it is equally important in the protection of traumatized lobules from the hazards of posttraumatic edema.

The level of action for these mechanisms that integrate ventilation and alveolar air tension, and that I suggest are available for edema control, seems to be at the bronchiole and respiratory bronchiole, the site of the sphincters around the necks of the air sacs. The character of the epithelium has already changed before this point is reached and goblet cells have disappeared. At the level of the segmental bronchus, however, the secreting membrane of the respiratory tract is well developed and introduces a factor in obstruction of the air passages that does not occur at the bronchiolar level. Obstruction of the segmental bronchus is followed by retrograde filling or drowning of the bronchioles and air sacs by the pent-up secretions. In the absence of infection this appears to be a relatively benign event. Accurate observations are not available, but presumably water and salts may be removed by the blood capillaries and protein by the lymphatic channels. The lumens of these obstructed small bronchi become filled by cylindrical casts of a tough rubbery substance. The situation is revealed in the pathologic findings of mucoceles and certain cysts but will not be explored at this time. However, certain physiologic and pathologic implications of the secretory activity of the bronchial mucous membrane deserve brief comment if only to display our present state of ignorance concerning them.

As an example, consider the enigma of the so-called Kartagener syndrome—bronchiectasis, situs inversus, and agenesis of the accessory sinuses. The pathogenesis of the bronchiectasis component has always been baffling. When Adams and I[5] tried to develop a hypothesis some years ago we could only postulate as others had done, that the bronchiectasis was a stigma of maldevelopment. Whether the maldevelopment was structural or took the form of some obscure predisposition to bronchiectasis could not be determined. It was pointed out, however, that the bronchiectasis itself took the form of the acquired rather than the congenital type. Since that time I have had the opportunity of observing two children with situs inversus and agenesis of the sinuses but without bronchiectasis demonstrable in complete series of bronchograms. Both children displayed an extraordinary increase in the secretory activity of the mucous membrane of the respiratory tract. This was manifested by the production of large quantities of mucous secretions both by cough and profuse nasal discharge. The content of pus was minimal. Both children had been born in the chemotherapeutic era of medicine and both had enjoyed the protective concern of well-to-do families able to provide winters in the southwest under able medical surveillance. Threatened episodes of respiratory infection had been handled by preventive measures including chemotherapy and careful observation for collapse and re-expansion of segments. The stage was set for the development of segmental or lobar bronchiectasis but as yet it had not appeared.

In the light of these observations a careful review of the childhood experience of an intelligent young man with the Kartagener syndrome, including bilateral basal bronchiectasis, brought forth a reconstruction of events that paralleled the story of the two children. The profuse mucoid secretion in his case became purulent only after an attack of bronchitis at the age of 14.

It seems, therefore, that the bronchiectasis of the Kartagener trilogy is a secondary manifestation and that the underlying disturbance may be an altered secretory activity of the respiratory tract epithelium.

Recently Bergstrom, Cook, and Scannell[9] have assembled a family history that not only confirms but throws added light on the subject. In this family the bronchiectasis, or as has been proposed the abnormality of the respiratory tract epithelium that precedes it, occurs in two siblings with situs inversus and in two others without situs inversus. Whether the stigma that affects the mucous membrane has a structural basis, a metabolic basis, or is attributable to some derangement of the neurosecretory mechanism, remains unknown. The question as to whether the respiratory mucous membrane behaves as a single organ in its secretory activity or whether lobar or segmental regional autonomy of control may exist also remains unanswered.

Returning to the pulmonary circulation, I remind you that many studies on minor pulmonary embolism that are now classics have demonstrated a collateral drift of blood in the pulmonary capillary network sufficient to maintain a slowly moving circulation despite occlusion of sizable terminals of the pulmonary artery. Increased back pressure in the pulmonary veins or abnormal slowing of the capillary stream by reduced respiratory excursion may convert a bland infarct into a hemorrhagic infarct. It is likely that this capillary blood drift crosses segmental boundaries. In the immediate situation imposed by the embolism, this drift of blood has no respiratory significance; having passed through functioning alveoli, the drifting blood is arterialized and not mixed venous blood. It may help maintain the integrity of the capillary wall, although there is some appearance of edema even in bland infarction, despite the fact that alveolar ventilation continues.

With the vast margin of safety provided the respiratory organs, the functional loss of a few lobules or even segments might seem to be unimportant. There is some evidence, however, to show that the lodgment of minute emboli of cells and agglomerations of tissue debris in the lesser circulation may be such a common event that it can be regarded as a physiologic happening. If this is so, the margin of safety would soon be used up if minor embolism in the terminal arteries always spelled hemorrhagic infarction. The healing process would reduce such an occluded area to a thin plate of fibrous tissue. A series of healed hemorrhagic infarctions thus would cause the remainder of the pulmonary tissue to distend with the changes of complementary emphysema and a concomitant reduction in functional capacity. The limited damage that attends bland infarction, in contrast, does not reduce the volume of the lung.

While the loss of a few square meters of diffusion surface from the 140 with which the lung is equipped for its primary function of respiration might be insignificant, the lung appears designed to conserve a *space-occupying* function. This is true even though the respiratory function of a segment is destroyed. It is observed in the series of events that follows bland infarction with the collateral drift of capillary blood. The same preservation of lung volume apparently to occupy space is also provided for by collateral air drift, as will now be shown.

After the demonstration of Van Allen and Lindskog[7] it was expected to find many instances where collateral respiration came into play in conjunction with pathologic lesions of the lung. However, both the original experiments and the postulated situations in which the drift of air might serve a useful function seemed to characterize collateral air drift as an emergency device. The reason it is not seen more commonly is that segmental bronchial obstruction at levels chosen for experimental demonstration and as observed with tumors, strictures, and foreign bodies takes place in the secretory zone of the respiratory tract epithelium. As a result, maintenance of air in the affected alveoli by passage through collateral channels soon is masked by the drowning of those alveoli with retained secretions. One must descend below the mucous cells to the finer ramifications of the air passages to find an example of collateral air drift at play. The example I shall provide is not protective in the sense of maintaining gas exchange function; it does act to conserve what I have referred to as the space-occupying function of pulmonary tissue that protects the remainder of the lung from complementary emphysema.

In describing the techniques of resection of a pulmonary segment,[8] reference was made to identification of the boundaries of a segment by deflating the lobe, clamping the segmental bronchus, and then inflating the lobe. The segment remains deflated. This is readily demonstrated in a normal lung and holds true with certain forms of bronchiectasis. After further study, however, it was apparent that only in the exceptional cases can this procedure be utilized. Either the segment under consideration is airless and shows the fibrotic changes of long-standing inflammation that preclude inflation, or the segment does *not* deflate when the endobronchial air pressure is reduced. Indeed, certain of these segments do not deflate when removed from the body and left with widely open bronchial apertures. The air in the alveoli is trapped air. It then occurred to us that bronchography not infrequently shows dilated bronchi extending into air-containing lung segments, but never with lipiodol entering the atria and alveoli. The dilated bronchi resemble the branches of a sea sponge known as "dead-man's-fingers" *(Chalina arbuscula)* and extend into a zone of radiant, air-containing parenchyma but in no way communicate with it. Other observers had commented on this phenomenon and referred to such bronchi as "leafless trees." Robert Klopstock was working with me at the time these observations were made, and using the techniques of Van Allen and Lindskog it was not difficult to demonstrate that the imprisoned air entered or escaped from the area either by way of an adjacent segment with normal bronchi or from an occasional normal branch bronchus within the segment. By more careful preoperative fluoroscopic examination it was possible to confirm the fact that the air in such a segment was indeed trapped exactly as in obstructive emphysema. From the standpoint of gas exchange, this area of lung is devoid of function. The air that reaches the alveoli within it has already passed through other alveoli and reached equilibrium with the pulmonary capillary blood. The air-containing segments are simply filling space. But this, as has been suggested, may have a functional significance.

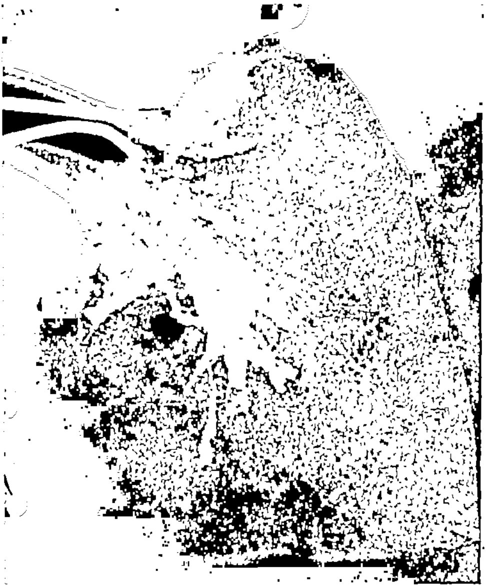


Fig. 4.

Klopstock studied these surgically removed lobes with lipiodol injections of the bronchi, correlating the observations with those made at operation and those made with air-flow techniques. Brief reference is made to three examples, using soft tissue roentgenograms as records.

Fig. 4 records the lipiodol injection of the air passages of a lobe excised from a 23-year-old man with bronchiectasis dating to attacks of pneumonia in early childhood. When the chest was opened the right lower lobe appeared emphysematous but with little or no deformity in its architecture. The dorsal segment deflated when the endotracheal pressure was reduced and inflated when it was elevated. The basal segments could not be deflated. The air that filled their alveoli was trapped, having entered through collateral channels by way of the dorsal segment. The lipiodol filling showed no communication between the dilated bronchi of the basal segments and the surrounding alveoli of those segments.

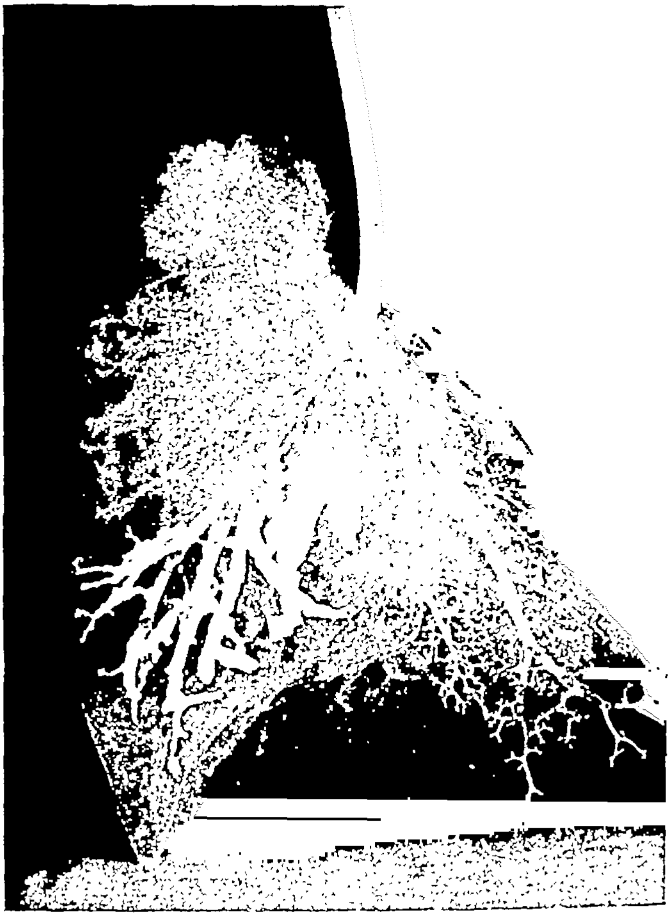


Fig. 5.

This specimen showed, in addition, a phenomenon that is quite constant and that provides further confirmation. The dorsal segment displayed the usual pattern of anthracotic pigmentation, but this was entirely lacking in the basal segments. It is understandable that airborne particulate matter cannot traverse the tortuous channels of the air-drift filter.

As an example of a more complex situation, reference is made to the lower lobe of a young adult woman (Fig. 5). The dorsal segment is not injected in this surgical specimen. *It still contains a cloud of alveolar lipiodol from the preoperative bronchogram showing normal air sac filling.* One of the three basal segments appears normal, one is grossly bronchiectatic, and one shows a cylindrical enlargement of the bronchi. In only one or two areas of the segment containing the cylindrically dilated bronchi do flecks of alveolar filling appear. All of these *segments were found symmetrically distended with air when exposed* at operation, in fact, architecturally the lobe appeared completely normal. The affected segments were being maintained in an inflated form by collateral air drift from the adjacent normal segments.

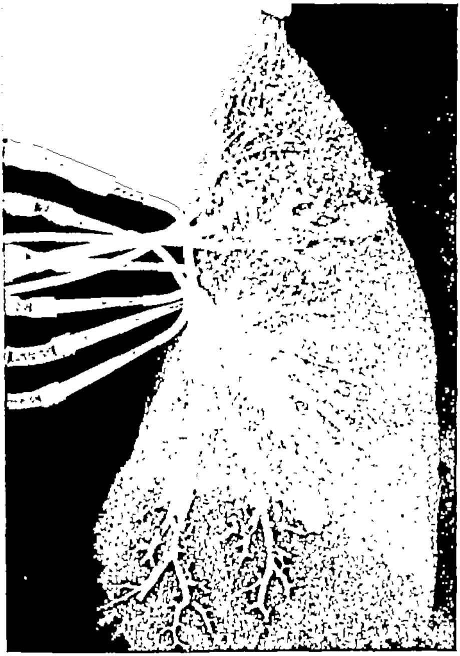


Fig. 6.

From the gross bronchiectasis displayed in these specimens, one may proceed to the state of affairs in the lobe shown in Fig. 6. This is the surgical specimen from an older woman, aged 56 years. Two bouts of pneumonia had been localized to this lobe, then *ensued cough, continuing slight fever, lassitude, and disability.* This patient was treated for many months with rest, chemotherapy, and climatic change but without improvement. The dorsal segment shows normal alveolar filling. In the basal segments there are a few areas in which the lipiodol enters the alveoli, but in general it is the picture of the "leafless tree," particularly in the mesial segment. There is little deformity that can be termed dilatation of the bronchi, and in fact it is a misnomer to classify this disease as bronchiectasis. The significant lesion appears to be *obliterative bronchitis* and *bronchiolitis.*

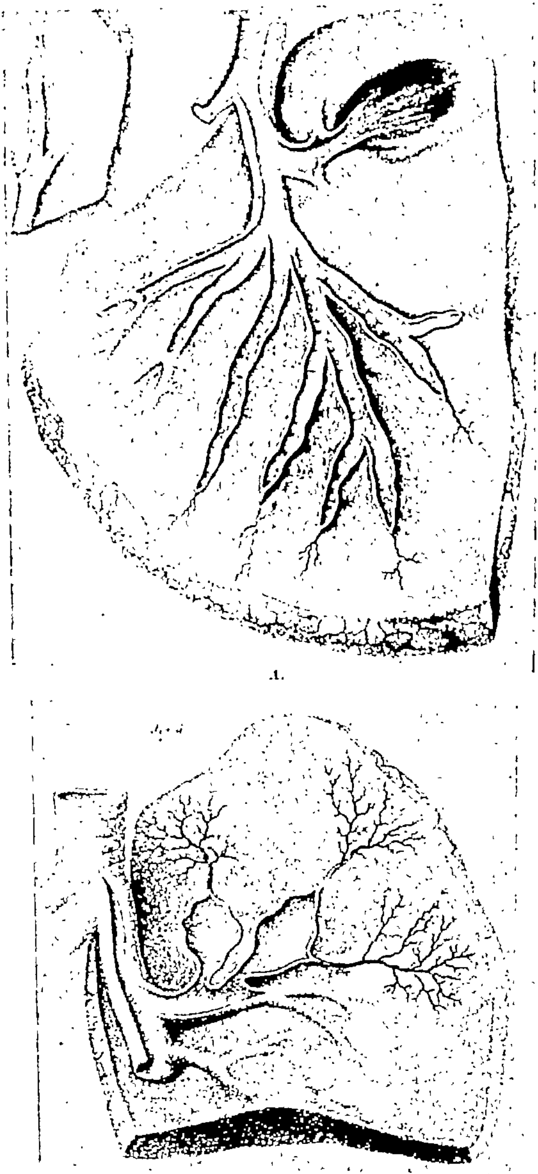
As shown in the preceding illustration this process may exist in one segment and true bronchiectasis in another. In some cases, as those described by Blades[9] as pseudobronchiectasis following virus infection, the obliteration appears to be temporary; in others, when the finer air passages are converted into fibrous cords, it is permanent.

For an adequate description of the morbid anatomy of the changes in the air passages that have been presented, one may go back to 1835.[10] This was the era when lungs were carefully examined in their three dimensional aspects and not chopped up into limp fragments for two dimensional cellular microscopy. In these old illustrations (Fig. 7) of bronchiectatic lobes, the bronchioles and presumably the smaller bronchi are shown reduced to fibrous cords that can be traced to the proximity of the visceral pleura. Observations regarding the state of the surrounding alveoli were not recorded.

I cannot agree with Fleischner[11] that this represents a *preatelectatic* phase in the development of bronchiectasis, although this could be true if the obliterative fibrosis of the bronchi and bronchioles progressed to involve all segments of a lobe. It appears, on the contrary, to represent a static phase of what may initially have been an acute inflammatory process that destroyed and obliterated the finer air passages. It might actually represent a *postatelectatic* phase, if collateral air drift penetrates an area that was atelectatic during the acute phase of infection.

As I have implied, there are, of course, other forms of bronchiectasis in which small dilatations and herniations of the bronchial wall can be traced in the finest bronchial twigs and even in the bronchioles. There are other more common types where inflammation and fibrosis have destroyed the architecture of the air sacs and bronchioles, and converted the lung or a portion of it into an airless, solid organ in which the mechanism of collateral air drift has been forever lost.

Obliterative bronchitis and bronchiolitis with or without atelectasis may be significant in the development of certain types of bronchiectasis and not merely be a by-product of the pathologic process. As a primary lesion it would favor the retention of secretion in the larger bronchi and possibly promote the atrophy of disuse. Basic to an understanding of obliterative disease in the finer air passages is a recognition that their lining membranes are very different from those of the larger divisions. They have a serous rather than mucous secretion. Thus, it is possible for the flames of infection to sweep across the lung and produce permanently obliterated bronchioles without evidence of collapse or fibrosis that is discernible on the x-ray film. Recent studies on *lung function* tend to confirm this statement.



A.

B.

Fig. 7.

Both capillary blood drift and interalveolar air drift are phenomena unique to this organ. Respiratory function depends on ventilation, not on alveoli containing air; it depends on an active capillary circulation, not on capillaries containing blood. Air-containing lung with normal pulmonary circulation as well as blood-containing lung with normal ventilation may be ineffective from the standpoint of respiratory exchange when the air has entered by way of adjacent alveoli and the blood has entered after traversing adjacent capillaries.

The structure of the lung appears to maintain its spatial integrity despite serious functional deterioration. Intricate mechanisms exist that act to maintain its volume and shape. It is unlike parenchymal organs that dwindle in size following the obliteration of ductal systems or occlusion of vascular channels. It does not become small with the changes of senile atrophy, in fact, the atrophic changes of the aging process produce quite the opposite result as recognized in senile emphysema. When normal lung is called upon to fill spatial demands the changes of complementary emphysema ensue, presumably with further reduction of the functional margin of safety with which this organ is so liberally provided. A segment of the kidney or the liver may be removed without significant reduction of the function of the part that is left behind. Not so with the lung. Surgical principles of conservation of function thus center in conservation of volume.

The apparent homogeneity of the structure and function of the lungs is highly deceptive. They are not two gas bags suspended in a container. Morphologically and functionally the lungs are a highly integrated organ system in which the whole must be considered in relation to the parts, and the parts in relation to the whole and to each other. The two lungs, the single lung, the lobes, the bronchopulmonary segment, and the lobules must be studied from this point of view. Only in this manner can the natural laws be disclosed that will ultimately guide pulmonary surgery.

I have made full use of the immunity that a Presidential Address affords to rebuttal and critical discussion. It must be taken for granted that the interrelationships of factual observations that have been presented will be recognized as hypotheses and valued only as such. I shall conclude with the expression of a conviction that is neither a fact nor an hypothesis, but a value judgment. Surgeons who stand on the threshold of the surgery of the lung today will not find prestige and fame in treading the easy pathways that were open to the older members of this Association. We enjoyed for a brief period the heyday of rapid technical development that follows the initial conquest of a new territory. Advance in the surgery of the lung will not be derived from technical experience alone. Anyone who gains the opportunity to operate on enough patients will have had experience, but it does not follow that one learns from it.

The area I have outlined is not only unexplored territory but it is a no man's land. Our allies in the medical sciences seem to have other pressing affairs that occupy their attention today. It is a territory in which surgeons may work but in which every gain must be bitterly fought for; it is remote from the glamour of scialytic lights and the comforts of lucrative trade. Yet in this area and adjacent ones lie the concepts that are essential if further advance in the surgery of the lung is to be realized.

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INTRATHORACIC MENINGOCELE

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ALTHOUGH, with increasing experience in dealing with mediastinal tumors, thoracic surgeons are becoming more accurate in their pre-operative diagnoses as to cell type, there is still an element of surprise when the tumor is exposed at operation. Among those tumors which present a degree of uniformity in the x-ray and clinical picture, and thus lend themselves to pre-operative diagnosis, are the intrathoracic neurofibromas. When a mediastinal tumor presents itself as a uniformly opaque, well-defined, rounded or lobulated mass in the posterior mediastinum, a high percentage will prove to be neurofibromas. When, added to this typical x-ray finding, there is the presence of bony absorption of adjacent posterior rib ends and the skin manifestations of neurofibromatosis of von Recklinghausen, one might feel justified in making the diagnosis of mediastinal neurofibroma. However, that even this typical picture may lead one into error is exemplified by the following case reports:

Case 1.—G. N., a 36-year-old white housewife, mother of four, was admitted to the University of Michigan Hospital on Feb. 23, 1947. She had been well until five years previously when she had been hospitalized, elsewhere, for "pneumonia and pleurisy." A chest x-ray examination of Oct. 27, 1941, revealed a 10 cm. tumor mass in the right apex posteriorly with a destructive process of the second and third vertebral bodies and the posterior aspect of the third rib. There was a sharp scoliosis to the left. A tentative diagnosis of neurofibroma had been made, and she was advised to have it removed. However, she did not seek further medical advice until one year prior to admission when she developed a persistent, nonproductive cough, mild, progressing to moderate, dyspnea and pain over the apex of the right chest.

The physical examination revealed generalized café-au-lait spots, most marked on the back, scoliosis of the upper dorsal spine with convexity to the right and dullness and diminished to absent breath sounds over the upper posterior right chest.

The laboratory studies revealed no abnormalities of the urine or blood. Roentgenograms revealed a huge lobular density of smooth outline occupying the upper two-thirds of the right hemithorax and situated posteriorly. There was deformity of the third rib, with apparent absorption of its posterior end and there were destructive changes of the second and third dorsal vertebrae.

A diagnosis of intrathoracic neurofibroma, possibly with intraspinal extension, was made and operation was performed on March 4, 1947, under intratracheal ether vapor anesthesia. The chest was entered posterolaterally through the bed of the fourth rib. A huge thin-walled cyst was found almost to fill the hemithorax. Aspiration, to facilitate mobilization, revealed clear watery fluid. It was felt that it might represent a pleural inclusion cyst similar to the so-called pericardial celomic cysts. However, further dissection revealed the cyst wall to be continuous with the dura through a defect measuring

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5 by 2 cm. in size, the defect arising as the result of enlargement and coalescence of the intervertebral foramina of D_2 and D_3, absorption of the pedicle of D_3 and erosion of the postero-lateral aspects of the bodies of D_2 and D_3. Through this defect could be seen the spinal cord and, higher in the spinal canal, at about the level of C_7, the inferior aspect of a small intraspinal cyst. The defect was closed by utilizing a portion of the cyst wall or

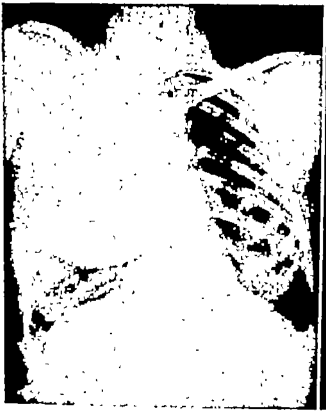

Fig. 1.

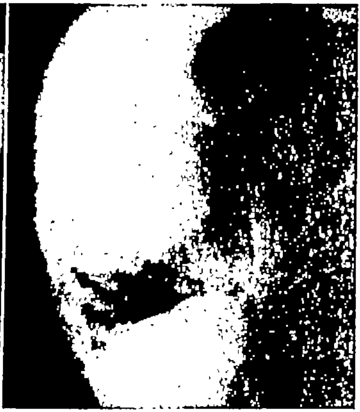

Fig. 2.

Fig. 1 (Case 1).—Preoperative x-ray film showing large intrathoracic mass, scoliosis, rib and vertebral changes.

Fig. 2 (Case 1).—Preoperative lateral film showing posterior location of mass.

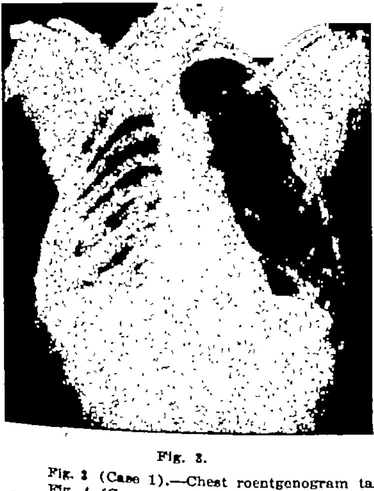

Fig. 3.

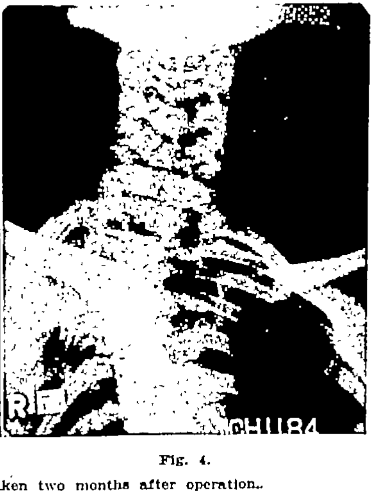

Fig. 4.

Fig. 3 (Case 1).—Chest roentgenogram taken two months after operation.

Fig. 4 (Case 1).—Detail film of apical region to show the upward displacement of rib 2, absorption of posterior end of rib 3, and the absence of rib 4 (excised at operation). The profound destructive changes of the bodies of D_2 and D_3 are shown.

dura as a flap. This was then covered by a free pleural graft and a layer of Gelfoam. Because the upper lobe had been partially collapsed for several years, it did not re-expand readily to fill completely the apex of the chest. An intercostal drainage tube was therefore employed for forty-eight hours.

Postoperatively, the patient experienced mild intermittent headache, relieved by aspirin from the fourth to the ninth days. Her temperature fluctuated from 98° to 102° F. for the first fifteen postoperative days. This was felt to be due to a slow seepage of spinal fluid into the chest. Following her discharge from the hospital, she continued to do well. At the time of her return visit on April 2, 1947, there were no complaints, her temperature had been normal, and the chest roentgenogram revealed that the pleural fluid had been largely absorbed.

CASE 2.—M. A. C., a 21-year-old white soldier had received a gunshot wound of the right arm in 1944, resulting in considerable disability of the arm. There was no injury of the chest or shoulder region. Upon being separated from the service Feb. 5, 1947, a routine chest roentgenogram was obtained which revealed a homogeneous round mass, about 2.5 cm. in diameter, in the apex of the right chest. He was admitted to the Percy Jones General Hospital. For diagnostic purposes a right pneumothorax was induced, revealing the mass to be extrapulmonary. A diagnosis of neurofibroma was made and on Feb. 24, 1947, this mass was explored through a posterior incision, resecting portions of the second and third ribs. The mass was revealed as a cyst, lying extrapleurally with its pedicle in the first interspace. The fluid could be easily expressed but it readily refilled. Ten cubic centimeters of clear fluid were aspirated. The cyst wall was relatively thick. Since it was considered to be a meningocele, was asymptomatic, and in a protected place, it was decided to make no effort to remove it.

Postoperatively, the patient had rather severe headache for several days, relieved by caffeine. After realizing that no demonstrable proof existed to prove this lesion a meningocele, a needle was inserted into the cyst, a small amount of spinal fluid withdrawn and 3 c.c. of Pantopaque injected. The isthmus was not seen fluoroscopically, but the Pantopaque readily entered the spinal canal. Detail films of the upper dorsal region failed to reveal any evidence of vertebral absorption or enlargement of intervertebral foramina.

CASE 3.—J. C., a 61-year-old white man, was admitted to the Letterman General Hospital on June 30, 1947, complaining of severe pain in the cervical region. Intermittent soreness and stiffness of the mid-dorsal region, relieved somewhat by physiotherapy, had been present for about five years. In May, 1946, dull aching pain began to occur in the cervical region. This discomfort was aided by physiotherapy, but one month prior to admission the pain was aggravated by massage. The physical examination revealed many small, soft nodules over the face, arms, legs, and body. Excision of one of these lesions revealed it to be a neurofibroma. There was café-au-lait pigmentation over the flanks and back. The neck was held in flexion with limitation of motion due to pain. A soft mass was present in the right cervical region and this had been present, unchanged, for several years. There was kyphosis of the cervical and upper dorsal spine with scoliosis to the left. Generalized arteriosclerosis was present, and there was a soft apical systolic murmur. The laboratory determinations were not abnormal. Roentgenograms revealed a rounded, well-circumscribed area of increased density, measuring 7 cm. in diameter, situated posteriorly in the right apex. This had remained unchanged for at least two years. No pulsation was present on fluoroscopic examination. The first, second, and third ribs were considerably eroded in their posterior portions with destruction of the heads of the first and second ribs. The first rib was disarticulated and pushed well forward of the transverse process. The right lateral aspect of the body of D_1 and the pedicles of D_1 and D_2 were destroyed. The intervertebral foramina of D_1 and D_2 were enlarged and apparently coalesced. A diagnosis of disseminated neurofibromatosis was made and the mediastinal mass was thought to be an intrathoracic neurofibroma. On Aug. 8, 1947, a right posterolateral thoracotomy was performed, the pleural cavity being entered through the bed of the resected sixth rib. A few adhesions between the mesial portion of the apex and the mediastinal mass were present.

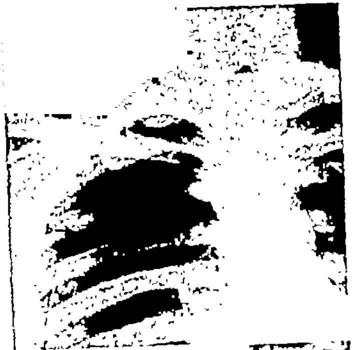

Fig. 5.

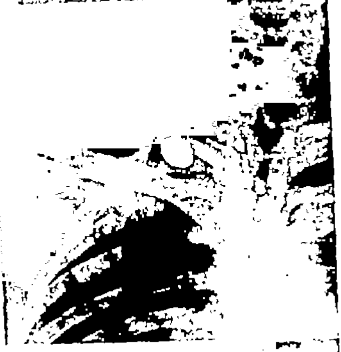

Fig. 6.

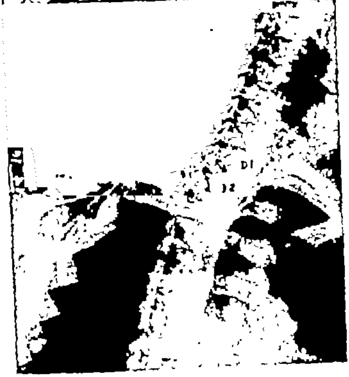

Fig. 7.

Fig. 5 (Case 2).—Film taken after induced pneumothorax. *a* indicates extrapulmonary mass, and *b* the collapsed lung margin. (Figs. 5, 6, and 7 from Sengpiel, G. W., Ruzicka, F. F., and Lodmell, E. A.: Lateral Intrathoracic Meningocele, Radiology 50: 515-520, 1948.)

Fig. 6 (Case 2).—Postoperative film revealing the presence of Pantopaque in the spinal canal following its injection into the cyst.

Fig. 7 (Case 2).—Film taken to outline intervertebral foramina. No enlargement is revealed.

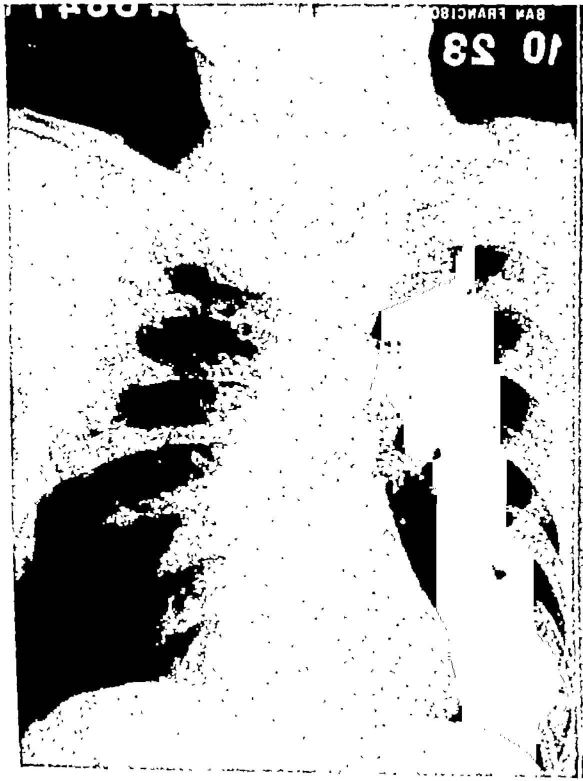

Fig. 8.

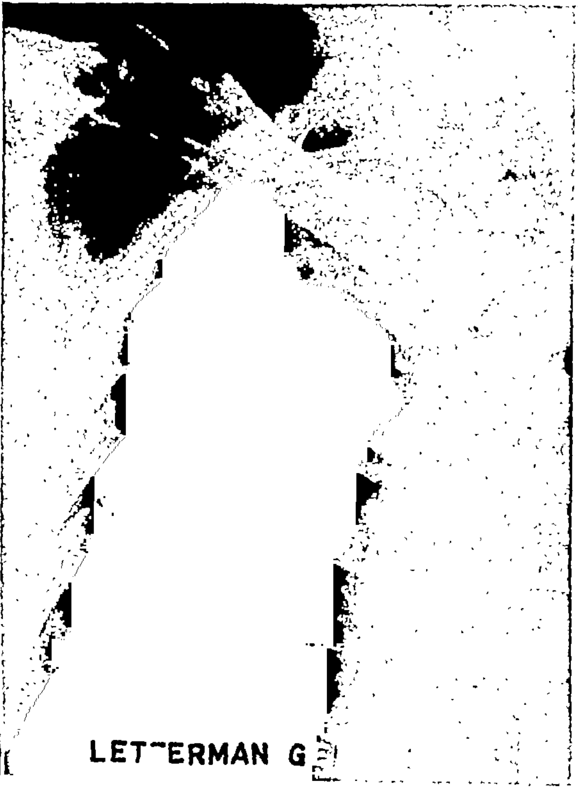

Fig. 9.

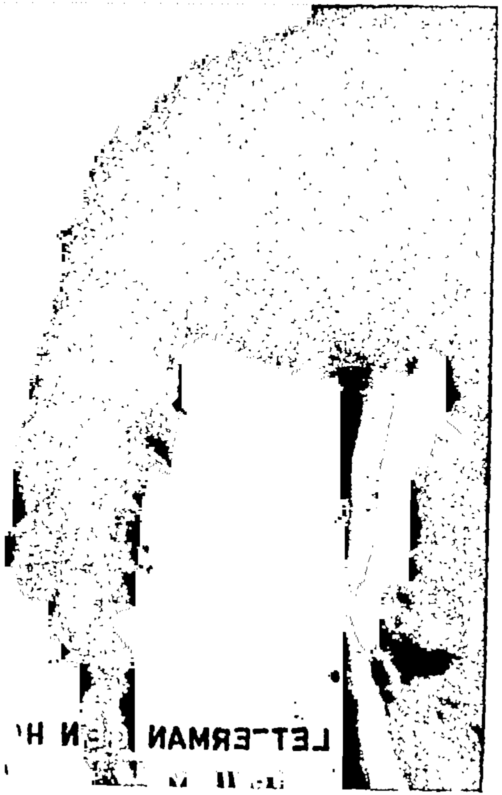

Fig. 10.

Fig. 8 (Case 3).—Frontal projection, showing the round circumscribed density in the cupola of the left thorax, measuring 7 cm. in diameter.

Fig. 9 (Case 3).—Frontal projection, bone detail, showing upper thoracic scoliosis with convexity to left. The posterior portions of the first, second, and third ribs are eroded. The first rib is disarticulated and pushed well forward of the transverse process. The intervertebral foramina of D_1 and D_2 are enlarged.

Fig. 10 (Case 3).—Right lateral projection with contrast barium in esophagus. There is no displacement of esophagus. Posterior position of the rounded mass is shown.

The latter was obviously cystic. The highest intercostal vein was stretched over the cyst. Subpleural dissection, begun posteriorly, resulted in troublesome venous bleeding. Approximately 500 c.c. of blood were lost from this source. The blood pressure fell to 80 mm. Hg, systolic, but the pulse rate remained at 88. The mass was aspirated and colorless fluid obtained. Further dissection of the cyst resulted in recurrence of bleeding and after about ten minutes the pulse and blood pressure could not be obtained. Bleeding was controlled by packing. The heart was stimulated by massage, injection of Adrenalin, and the forced transfusion of 1,500 c.c. of whole blood. There was return of cardiac action, the blood pressure mounting to 100 mm. Hg systolic. Further attempts to enucleate the cyst revealed that, superiorly, portions of the brachial plexus were stretched over the cyst. Fifteen minutes later there was a sudden drop in blood pressure and cardiac arrest. No response occurred to injection of Adrenalin and massage through the opened pericardium.

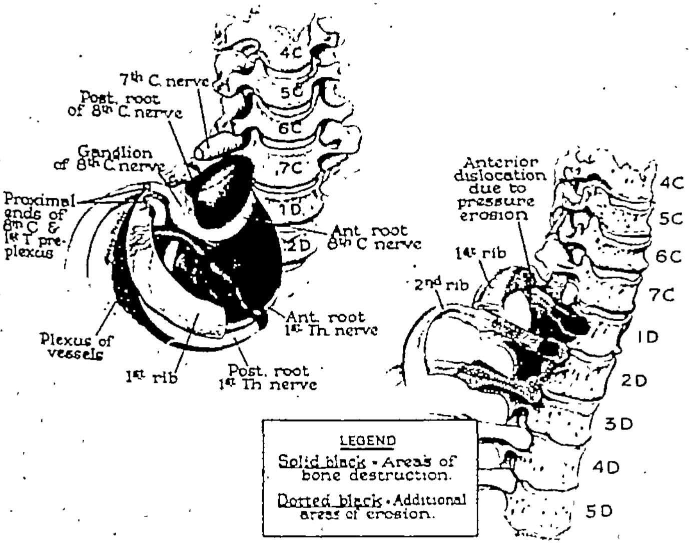
4C
5C
6C
7C
1D
2D
7th C nerve
Post. root of 8th C nerve
Ganglion of 8th C nerve
Proximal ends of 8th C & 1st T pre-plexus
Ant. root 8th C nerve
Ant. root 1st Th nerve
Plexus of vessels
1st rib
Post. root 1st Th nerve
Anterior dislocation due to pressure erosion
1st rib
2nd rib
4C
5C
6C
7C
1D
2D
3D
4D
5D
LEGEND
Solid black - Areas of bone destruction.
Dotted black - Additional areas of erosion.

Fig. 11 (Case 3).—This drawing demonstrates the relationship of the meningocele to first and second ribs, and to the lower trunk of the brachial plexus. The figure to the right shows the extent of bone erosion and bone destruction.

Postmortem examination revealed a tissue-thin meningeal sac projecting extrapleurally through the enlarged intervertebral foramen of C_7. The spinal cord was visible through the opened sac, the wall of which was intimately adherent to the vertebral bodies and the eroded first rib. The spinal cord in this region was diffusely thickened and whitish.*

In 1933, Pohl[1] reported a case of neurofibromatosis with intrathoracic meningocele. The meningocele was excised, the patient dying of empyema. Schuller and Uiberall[2] reported the case of a 56-year-old man who, in 1926, had a diagnosis of neurofibromatosis with intrathoracic tumor. In 1935, the tumor at the level of D_7 to D_9 was described as the size of an apple. Lumbar puncture revealed fluid under normal pressure with a positive Pandy and slight xanthochromia. Lipiodal was injected into the spinal canal and found to enter the tumor. There was evidence of spinal block at D_8. A neurectomy was performed to relieve his pain, but he died following this procedure. The autopsy showed a 10 cm. cyst, absorption of the dorsal surfaces of vertebral bodies D_7 to D_{10}, and an intervertebral foramen large enough to admit two fingers. They had not felt justified in attacking the cyst as Naffziger had warned of the danger of hydrothorax due to drainage of spinal fluid and they further felt that closure of the defect might be difficult. They stress the value of myelography in suspected cases.

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*Complete details of the neuropathologic findings, not germane to this presentation, are being reported elsewhere by Dr. W. H. Garol, Neurosurgical Consultant, and Major H. Sprinz, Chief of Pathology Section, Letterman General Hospital.

Ameuille, Wilmoth, and Kudelski[3] induced a diagnostic pneumothorax in a 46-year-old woman with a large posteriorly located mediastinal tumor. As a further diagnostic procedure, a thoracoscopy was performed and this revealed a large cyst which was translucent, reddish-white, and with numerous vascular arborizations. A diagnosis of hydatid cyst was made and thoracotomy performed. The cyst was found to contain clear fluid. A defect was found in the lateral faces of D_8 and D_9 which seemed to lead into the spinal canal although the spinal cord was not seen. It was not possible to approximate the pleura over the defect. Postoperatively, the patient developed fever, abdominal ascites, urinary retention, and fecal incontinence. The spinal fluid was found to contain staphylococci, and she died on the eighth postoperative day. At autopsy, the chest was found to contain 2,000 c.c. of diluted syrup-colored fluid. The lesion was found to be a dural sac escaping through the defect in the bodies of D_8 and D_9.

Campos[4] reported two cases of what probably represent intrathoracic meningocele, although anatomical confirmation by operation or autopsy is lacking. One was a Brazilian boy, aged 7 years, with the presenting complaint of dorsal scoliosis. X-ray examination revealed a large pedunculated shadow, 5 cm. in diameter, with well-defined margins in the upper right chest. The first six dorsal vertebrae were bifid and there was synostosis of the fourth and fifth dorsal vertebrae. Injection of opaque medium was not employed. The other case was of a 54-year-old Japanese man with symptoms of pericardial pain, dyspnea, and nonproductive cough. A diagnosis of neurofibromatosis was made and confirmed by biopsy of a skin nodule. The chest roentgenogram showed a rounded, well-defined shadow in the right upper chest far posteriorly and an increase in size of the third, fourth, and fifth intervertebral foramina. Penetration was described as occurring through the fifth foramen.

The significance of the fact that neurofibromatosis was present in three of these five reported cases is not clear. Case 1 of this report had café-au-lait spots but no neurofibromas and Case 3 had both. Certainly, it is not of diagnostic aid in differentiating between intrathoracic neurofibroma and intrathoracic meningocele, and, in fact, adds considerable confusion, since one might expect mediastinal neurofibroma as part of the generalized syndrome. Similarly, the rib changes, intervertebral foraminal enlargement, and vertebral body destruction may, and frequently do, occur as a result of neurofibroma. Short of thoracoscopy or thoracotomy, therefore, the only procedure that offers a good chance of differential diagnosis is the intraspinal injection of an opaque medium and positioning of the patient so as to fill, if possible, the suspected meningocele.

The demonstration of a spinal fluid block above the level of the meningocele in the case of Schuller and Uiberall would seem to indicate the presence of an intraspinal lesion, and it will be recalled that in Case 1 of this series a small intraspinal cyst was observed. The simplest explanation of the production of the meningocele itself would seem to be that there is herniation of the dural envelope of a spinal nerve through the intervertebral foramen with pressure necrosis of the vertebral bodies and enlargement of the foramen as the meningocele enlarges. This, however, does not explain the presence of the intraspinal cyst. It seems possible that both the meningocele and intraspinal cysts could have arisen at points of congenital weakness of the dura as in the spinal extradural cysts described by Cloward and Bucy.[5]

Once the diagnosis of intrathoracic meningocele is made, either by demonstrating a communication with the spinal canal or by exploration, the question of the advisability of operation arises. The two patients reported upon by Pohl, Ameuille, and associates, both died of empyema and meningitis, without, of course, the benefit of chemotherapy. In both cases, difficulty was encountered in closing the defect. In our Case 1, a satisfactory closure was also difficult but fortunately seems to have been attained. The dura at the neck of the sac was very thin and of little tensile strength, probably due to stretching by the weight of the cyst. Patients in whom the meningocele has attained such size, as in Case 1 of this report, that respiration is either seriously interfered with, or potentially embarrassed, will require at least partial excision of the meningocele. It would seem advisable to save a generous portion of the neck of the sac so that a plicated closure can be obtained. It is possible that the use of bone grafts to bridge the vertebral or foraminal defect may be feasible. In patients with relatively small sacs, operation is probably not justified unless serial chest roentgenograms reveal progressive enlargement to the point of progressive respiratory embarrassment.

SUMMARY

Three cases of proved intrathoracic meningocele are presented, in one of which excision of the sac with good early recovery was performed.

The literature is reviewed, five previously reported cases being discussed. The two patients in this series on whom excision of the sac was performed both died as a result of empyema and meningitis.

The chief diagnostic aid in differentiating between meningocele and neurofibroma is the intraspinal injection of opaque medium.

The question of advisability of excision of the sac is discussed.

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DISCUSSION

DR. HARVEY J. MENDELSOHN, Cleveland.—I wish to present an additional case of intrathoracic meningocele from Cleveland City Hospital. The patient had been picked up in a routine chest survey by the City Tuberculosis Clinic as having an intrathoracic tumor. She had been asymptomatic until she knew she had this tumor, and she was admitted to the hospital about five months later, at which time she said she had some dyspnea, but on observation in the ward appeared asymptomatic.

(Slides) This shows the tumor in the posteroanterior film, and this shows the posterior position in the lateral film. This patient also had generalized cutaneous neurofibromatosis. Our preoperative diagnosis was mediastinal neurofibroma. However, we suspected some intraspinal involvement. The spinal fluid studies were normal. The patient was explored in August and the defect in the spinal canal can be seen here. We closed it in the same way described by Dr. Byron, by imbricating the sac. We sprinkled some talc in the area. She had an uneventful recovery and was kept in bed for two weeks.

The film one year later showed a normal chest, and the patient is asymptomatic except for a complaint of low back pain.

DR. JEROME R. HEAD, Chicago.—I would like to report an additional case. I believe that the rarity of the condition warrants doing so. The patient was a man, aged 43 years. He entered Hines Veterans Hospital for some other complaint. A routine x-ray film of the chest revealed a globular tumor in the left lower thorax behind the heart. The tumor was directly adjacent to the spine. A needle was inserted into the tumor and crystalline-clear fluid was obtained. Following the aspiration of about 10 c.c. of fluid, the patient developed a typical postlumbar-puncture headache. Several days later a needle was reinserted into the tumor mass and at the same time a needle was inserted into the spinal canal. Withdrawing fluid from the tumor lowered the pressure in the spinal canal, and withdrawing fluid from the spinal canal lowered the pressure in the tumor. The specimens of fluid removed through each needle were comparable in gross appearance, in cell count, and in all chemical analyses. These tests seemed to warrant the diagnosis of intrathoracic meningocele. The patient was not operated upon.

During the preceding discussion concerning the surgery of these meningoceles, the question has been raised concerning the leakage of spinal fluid into the pleural cavity. The following case bears upon this possibility. Some months ago I exposed the stellate ganglion for a vascular surgeon in a case of Raynaud's disease. The exposure was performed in the same manner as an extrapleural pneumothorax. The vascular surgeon then avulsed the sympathetic roots, the rib bed was closed with a running suture, and the wound was closed without drainage. The patient developed a collection of fluid in the extrapleural space. On aspiration this was found to be spinal fluid. Following aspiration of the fluid, the patient developed headache and the fluid rapidly reaccumulated. The question arose as to what to do concerning this complication. We decided to do nothing. The patient had no symptoms referable to the condition and has remained well since.

DR. FRANK S. DOLLEY, Los Angeles.—There are two very important diagnostic symptoms in these cases that Dr. Byron has reported that should be emphasized.

Several years ago, Dr. Brewer and I had published a paper on intrathoracic tumors. In writing of the paravertebral tumors high in the cupola of the hemithorax, we stated that the so-called Pancoast tumor was, in a great majority of cases, actually a primary bronchiogenic carcinoma, having its origin close to the visceral pleura against the mediastinum. We showed that there are a number of other conditions which may cause this paravertebral or paramediastinal shadow, particularly fibromyxoma that would be operable and early fibromyxosarcoma that might also prove to be operable. We did not, however, mention meningocele. This manifestly should be included in this group.

The second point for emphasis is a question of bone erosion.

I have been accustomed to believe that any sign of bone erosion not due to aneurysm or cold abscess could be considered as representing a malignant process and was inoperable.

It is most interesting to me to learn that two of these cases of Dr. Byron's this morning show widespread evidence of bone erosion. The only explanation which occurs to me is that the same sort of mechanism is taking place in these cases as in aneurysm; that is, the meninges themselves do not readily stretch; and the increased pressure against the bone contiguous to the meninges in a situation that does not permit lateral expansion results in bone erosion. Therefore we certainly are not justified, if aneurysm or cold abscess can be eliminated, in looking upon the condition where bone erosion exists as surely malignant and inoperable.

DR. CIAGLIA, Utica, N. Y.—I had no intention of presenting this case but the features were so similar to the ones presented that I thought a few words on it might be in order. This patient was a 31-year-old white woman, who was minding her own business working in a mill, when someone did a survey, and a tumor was reported. She was completely asymptomatic. She also had clinically multiple neurofibromatosis, later proved by biopsy. She had also bony changes with a slight angular scoliosis in the upper thoracic region. The x-ray examination showed a mass clearly defined and rounded, about the size of an orange, in the left apical region. She was explored transpleurally and much to my surprise the tumor was fluctuant. A needle was put in and a crystal-clear fluid was obtained. Later, on examination of this fluid, it was typical spinal fluid with sugar and chlorides and so forth. The pedicle was ligated where it came from the vertebra in the region of the first and second. Similar plastic repair was done as has been described and the postoperative course was completely uneventful except for a Horner's syndrome of moderate degree.

The woman is back at work and I can see no sign of recurrence so far.

DR. BYRON.—This seems to be one of those entities that appear occasionally; one never hears about them and then one learns of a number of cases all together. For instance, pericardial celomic cysts were unheard of until Dr. Lambert reported a case in 1940. Now we know they are very common, and most of us have seen cases. Intrathoracic meningoceles may be reported with increasing frequency. I think there is some importance in making a preoperative diagnosis, because I do not believe operation is indicated in all cases. In the first place, with a large bony defect, it is questionable in many cases whether the defect can ever be satisfactorily closed, and second, since I believe that the meningocele is a herniation of the dura, I see no reason why herniation cannot recur.

THE USE OF TUBED PEDICLE GRAFTS IN CARCINOMA OF THE UPPER ESOPHAGUS

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BEN EISEMAN, M.D. (BY INVITATION)
ST. LOUIS, MO.

CANCER of the esophagus has long tested the resourcefulness of surgeons and despite a prodigious amount of work and no little ingenuity, the results until very recently have been far from noteworthy. However, the past decade has witnessed very significant successes in the conquest of this formidable disease.

North American surgeons and members of this association in particular have contributed pre-eminently to this progress. Adams and Phemister's[1] successful one-stage resection with esophagogastric anastomosis, the first on this continent, stimulated great interest. The subsequent significant contributions in this field by Eggers,[2] Churchill,[3] Garlock,[4] Ochsner and DeBakey,[5] Rienhoff,[6] and in particular Sweet,[7, 8] are too well known to this group to warrant review. Suffice it to say that few of experience would question the desirability of gastric substitution when esophageal sacrifice is necessary. The stomach has been carried higher and higher into the chest so that Sweet[9] has been able to report recently the very creditable achievement of anastomosing the stomach to the esophagus high in the neck.

Despite the eminently satisfactory results with the esophagogastric procedure in lesions from just above the aorta downward, there is a sizable group of the higher lesions for which in our opinion one-stage resection with esophagogastric anastomosis can never be the procedure of choice. Certain features of carcinoma of the cervical and upper thoracic esophagus appear to us of such importance as to justify the consideration of this lesion separately from carcinoma of the middle and lower third.

Rational cancer surgery unequivocally requires the removal of the entire gland-bearing area of the involved structure and the radical extirpation of the lesion. The requirements of a satisfactory cancer resection are well fulfilled for carcinoma at and below the level of the aortic arch by the procedure of resection and esophagogastrostomy in one stage. Lesions above this inevitably become cervicothoracic problems since from this area the lymph drainage is chiefly to the superior and inferior deep cervical lymph nodes (Fig. 1). This has been well shown by the studies of McGibbon,[10] Sakata,[11] and others. Submucosal infiltration, both upward and downward for great distances, occurs often in esophageal cancers so that one might question the validity of ever limiting the resection to the neck. It would appear illogical to limit the resection of the carcinoma and its regional lymphatics simply because a means of reestablishing the

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Read at the Twenty-eighth Annual Meeting of The American Association for Thoracic Surgery, Quebec, Canada, May 31, June 1, 2, 1948.

physiologic function is easily at hand. As great a boon as the one-stage resection with esophagogastrostomy has been, we doubt that its effectiveness can be extended consistently to lesions well above the arch or into the neck.

A further point of importance in our sector is that unfortunately a significant number of people present themselves with an all too frequently ill-done

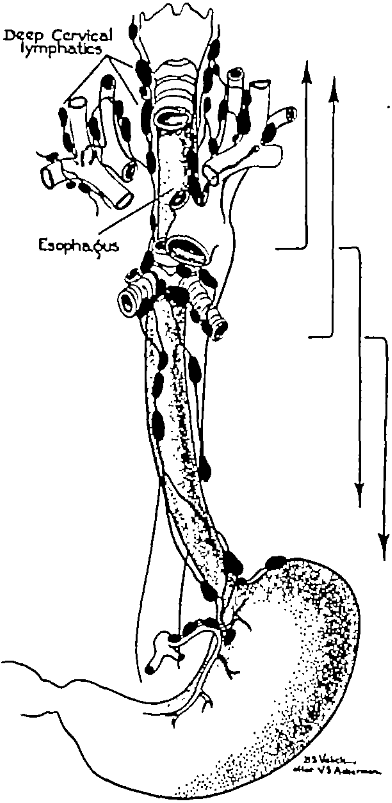

FIG. 1.—The lymph drainage of the upper third of the esophagus is predominantly to the lymph nodes in the lower cervical and supraclavicular region.

gastrostomy. The difficulty or impossibility of elevating such stomachs to the neck or even to the cupola of the thorax is obvious.

It would seem then that a combined cervicothoracic resection with radical resection of the regional cervical lymph nodes bilaterally en bloc would more nearly fulfill the requirements of a satisfactory cancer resection in carcinoma of the upper esophagus. Such a procedure, of course, implies staging, and in this time of enthusiasm for one-stage operations, one can anticipate the criticism leveled at staged procedures. However, this factor can be considered of no more than secondary importance since as Lilienthal[12] so well stated years ago, "The number of operations necessary to accomplish the desired result is of far less importance than its final success."

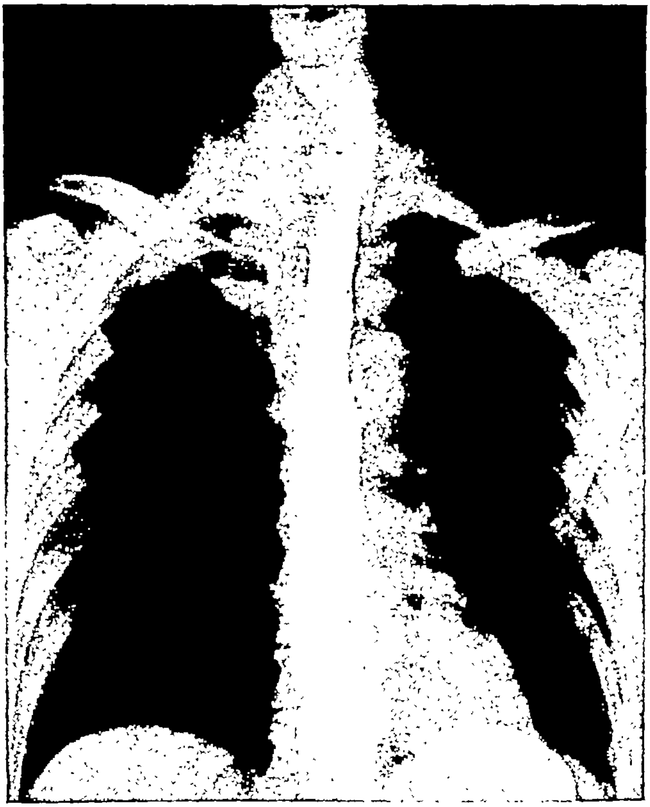

Fig. 2.—Case A. M., showing esophageal defect. The defect and distortion were due to the combined effects of a huge goiter and an annular carcinoma of the esophagus at the level of the first thoracic vertebra.

Struck by the success of Wookey[13] of Canada in the management of carcinoma of the esophagus confined to the upper cervical segment, we decided in 1946 to attempt the incorporation of the principles outlined here into a plan of management for high esophageal lesions. The problem was first approached in the laboratory in the summer of 1946. The first attempts were made toward resection of the esophagus of the dog and immediate replacement with full-thickness free skin grafts. Although free graft was accomplished, the procedure was considered to be of no practical value because of the subsequent contracture of the graft and production of an esophageal obstruction. Attention was then turned toward working out a technique of pedicle graft replacement of the upper portion of the esophagus using a tubed pedicle graft based on the neck and passed through the superior thoracic aperture into the chest for direct anastomosis to the esophagus. Technical difficulties were encountered in the dog which led to the impossibility of always obtaining primary healing and prevented carrying the procedure to completion in a single dog. However, it was learned that long pedicle grafts could be easily prepared and transplanted into the chest and that they could be anastomosed to the esophagus without stricture formation. The technique was developed to such an extent that it was felt justifiable to apply it to a suitable human case. Such a patient was found and is the subject of the following case report.

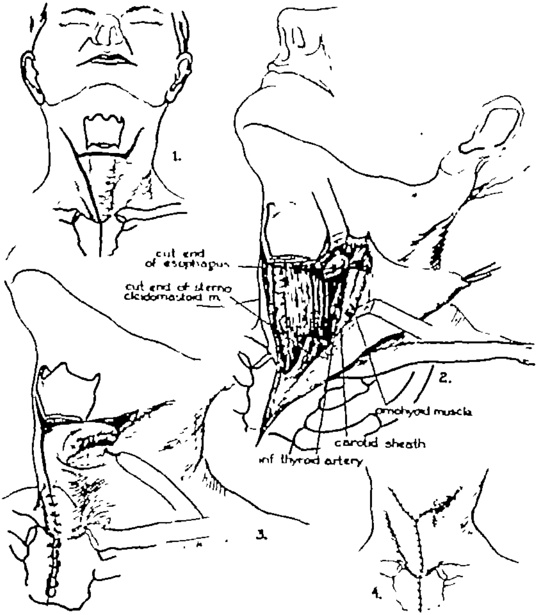

Fig. 3.—The diagrams illustrate the cervical portion of the first stage. This is a combined cervicothoracic procedure, the thoracic exposure being through the right third intercostal space (the same as illustrated in Fig. 6).

The cervical incisions (*1*) are made in such a way as to turn a triangular flap. The esophagus is amputated high above the lesion at the inferior border of the cricopharyngeus (*2*) and after the resection the triangular flap is sutured to the cut end of the esophagus (*3*) in such a manner as to complete the upper end of the esophageal reconstruction.

CASE REPORT

A. M., a 62-year-old white woman, was admitted to the Barnes Hospital on Sept. 2, 1947, with the complaint of difficulty in swallowing for the preceding three months. She had a sensation as if food stopped in the throat at the level of the sternal notch. This difficulty was progressive until at the time of admission she could swallow only liquids and various soft foods. The patient had had enlargement of the thyroid gland since 12 years of age. Ten years before admission she went through a period of nervousness, which was attributed to thyrotoxicosis, and which responded to iodine therapy. She felt that the thyroid gland was larger at the time of this admission than it had ever been before. There had been a weight loss of five pounds in the preceding three months.

The physical examination was not remarkable, except for the presence of the enlarged thyroid, which was about three times its normal size.

X-ray examination of the esophagus revealed displacement of the esophagus to the left and irregular filling, which was considered probably due to pressure from the enlarged thyroid gland. This filling defect started at about the level of the first thoracic vertebra (Fig. 2).

On September 6 an esophagoscopy was performed, at which time a lesion was encountered 18 cm. below the upper alveolar border. This was an annular mass, biopsy of which revealed squamous cell carcinoma, grade III.

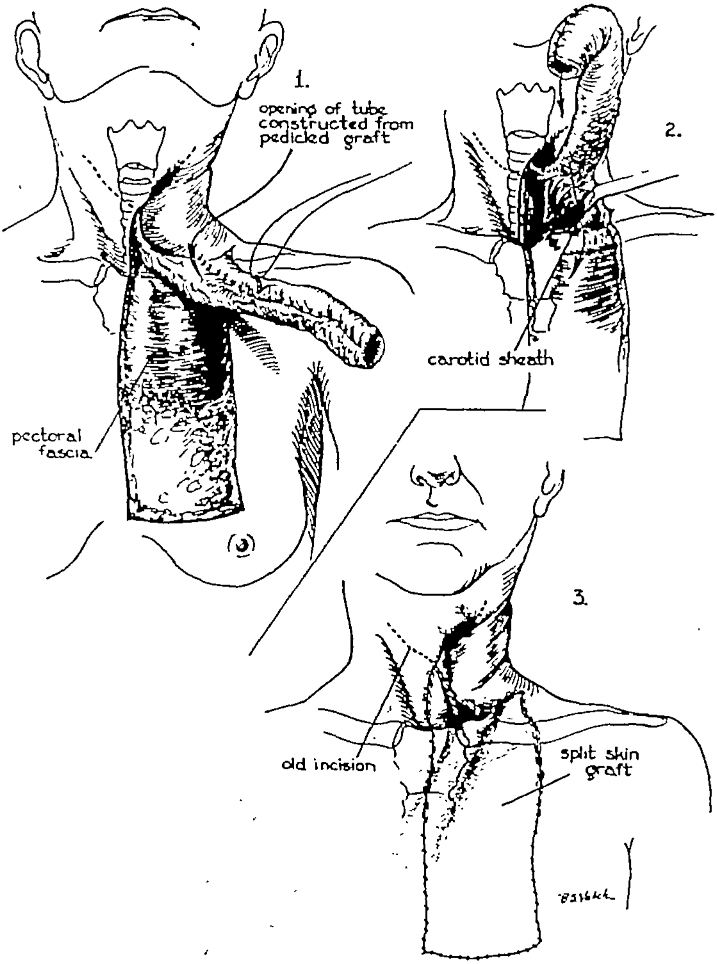

Fig. 4.—After the pedicle graft of the anterior chest wall has been prepared by the usual delaying procedures which insure its blood supply, the graft is converted into a tube (*1*) and passed into the chest medial to the left carotid sheath and posterior to the trachea (*2* and *3*). This procedure again requires a combined cervicothoracic exposure for anastomosis of the graft to the esophageal stump. The donor area from which the pedicle graft was removed is covered with a split-skin graft (*3*).

The patient was carefully evaluated and considered to be a good operative risk. There was no evidence of regional metastasis in the neck. It was decided that a resection of the upper third of the esophagus by a combined cervicothoracic procedure be done, to be followed by reconstruction of the esophagus at a later date. On Sept. 26, 1947, a combined cervicothoracic resection was performed. The right side of the chest was entered through the third intercostal space anteriorly. The lesion was identified with ease slightly above the level of the aortic arch. The esophagus was separated from the vagus nerve and from the trachea, the dissection being carried high into the neck. The tumor was readily palpable and dissected free from surrounding structures. The esophagus was sectioned just below the level of the aortic arch, and the lower end was closed with inverting sutures, then anchored to the prevertebral fascia in order to prevent its retraction downward. The upper end of the esophagus was closed with a ligature, following which the chest incision was approximated and a dressing applied while the attention was turned to the cervical portion of the operation. Incisions were then made in the neck in such a manner as completely to expose the neck anteriorly. A triangular flap was outlined by the incisions as illustrated (Fig. 3). Since the large goiter prevented access to the esophagus, a subtotal thyroidectomy was done, care being taken to protect the recurrent laryngeal nerves. The left sternocleidomastoid muscle was resected, and the contents of the left carotid sheath were retracted laterally. The esophagus was exposed and dissected free of its posterior attachments and of the trachea anteriorly. It was then delivered through the neck after being completely mobilized. The dissection of the esophagus was carried up to the inferior border of the cricopharyngeus muscle at which level it was sectioned. The triangular skin flap was then sutured to the remaining end of the esophagus, and the wound was closed as illustrated (Fig. 3). The thoracic wound was then closed after a catheter had been introduced into the right pleural space for water-seal drainage.

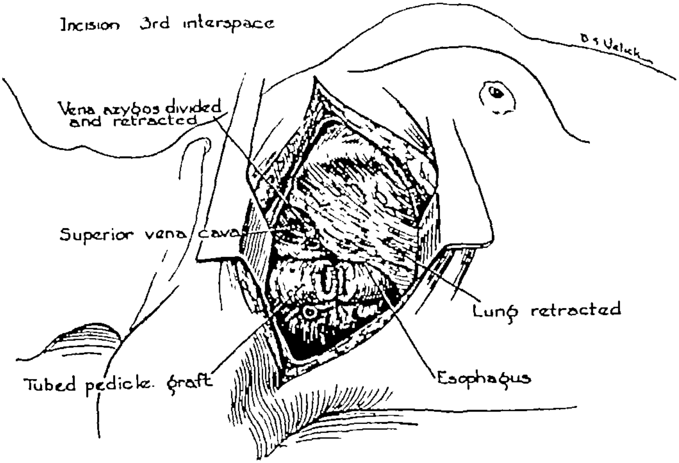

Fig. 5.—The right anterior thoracic approach is again used. It is of the greatest importance that the anastomosis be done meticulously and that perfect primary healing ensue if a stricture is to be prevented.

The postoperative course was uneventful. On Oct. 1, 1947, a Stamm type gastrostomy was done for feeding.

On October 15 the first stage operation for preparing a tubed pedicle graft from the anterior chest wall was performed. Six weeks later the pedicle graft was ready for transplantation into the chest, four minor preparatory procedures having been accomplished in the meantime.

On December 9 the pedicle graft from the anterior chest wall was converted into a tube and inserted into the chest through the superior thoracic aperture and anastomosed to the distal end of the esophagus. This again was a combined cervicothoracic procedure, the thoracic approach to the esophagus being through the right third intercostal space (Figs. 4 and 5). The defect on the anterior chest wall was covered with a split-skin graft.

During the operation biopsies were taken from the neck, from the posterior mediastinum, and from the distal esophageal stump. All of these biopsies subsequently showed what appeared to be a wildly growing anaplastic type of carcinoma.

In view of the positive biopsies, it was decided to give the patient radiation therapy before attempting to complete the esophageal reconstruction. This was done through four posterior thoracic portals, cross-firing the mediastinum; and through two cervical portals, which spared the area which it was hoped might later be used for esophageal reconstruction. The x-ray therapy was started on December 22 and discontinued on Jan. 17, 1948. During this period the patient was fed by a tube through the new esophagostomy opening in the neck, and the gastrostomy was allowed to close.

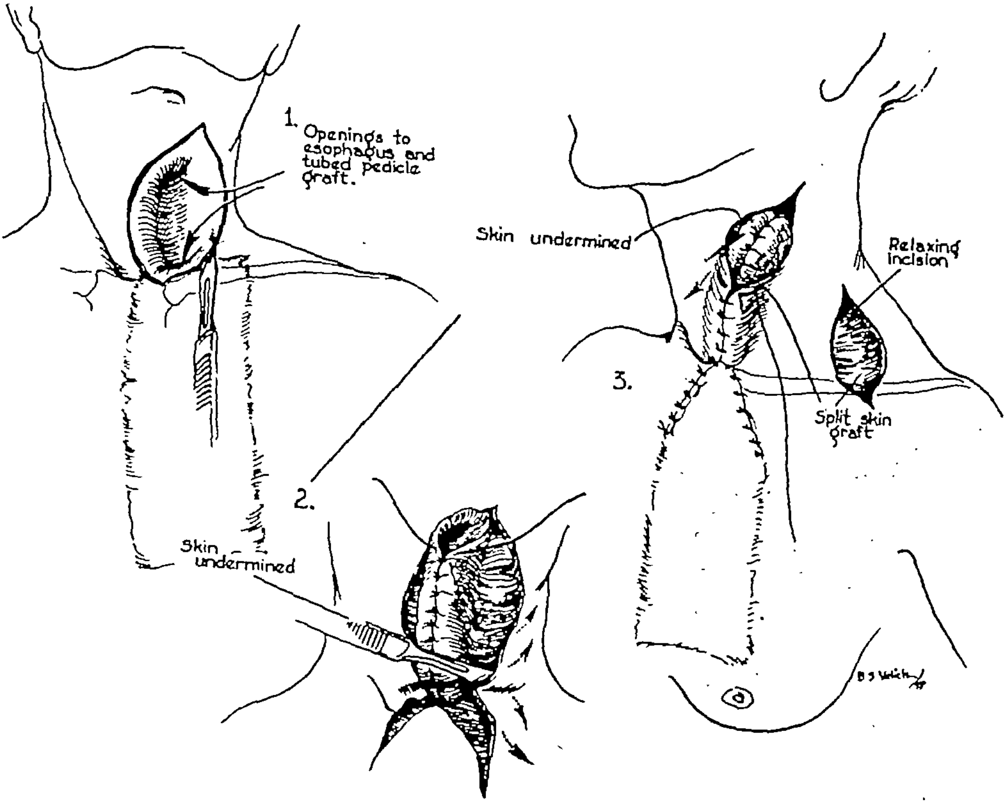

Fig. 6.—The final stage in the reconstruction consists of completing the tube construction in the neck. The upper end of the anastomosis done at the first stage (Fig. 3) is not disturbed. The tube is completed (*1* and *2*), following which the large superficial defect is closed by sliding the skin from both sides and using relaxing incisions as indicated (*3*).

Following subsidence of the radiation reaction, the patient's condition improved markedly, and on March 13, 1948, the cervical portion of the reconstruction was completed (Fig. 6). At the time of this operation some small nodules were encountered deep in the neck which subsequently proved to be carcinoma. The operation was completed in spite of this finding.

The postoperative course was uneventful. The patient was fed through an indwelling tube which had been passed through the nose and into the stomach at the last stage of the reconstruction. This tube was removed on the third postoperative day. Three days later the patient was started on water and liquids by mouth. She could swallow with ease. On the ninth postoperative day she was placed on a soft diet of puréed vegetables, chopped meat, etc., which she took without difficulty. This was rapidly advanced to a regular diet. By the end of two weeks the patient was taking the same diet as other unrestricted patients on the ward. In view of the positive biopsies at the time of the last operation, it was decided again

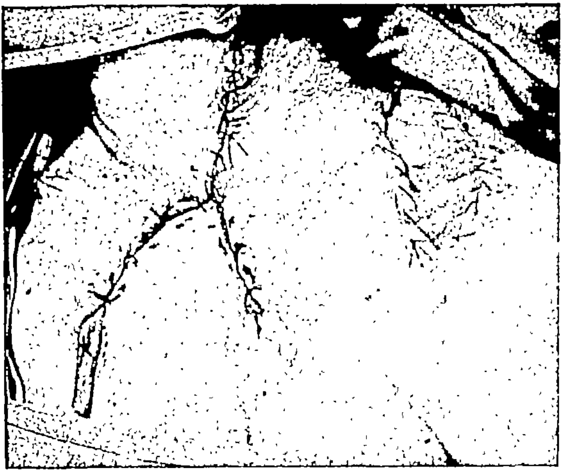

Fig. 7.—The cervical tube is completed and the defect closed. The relaxing incision has been covered with a split-skin graft.

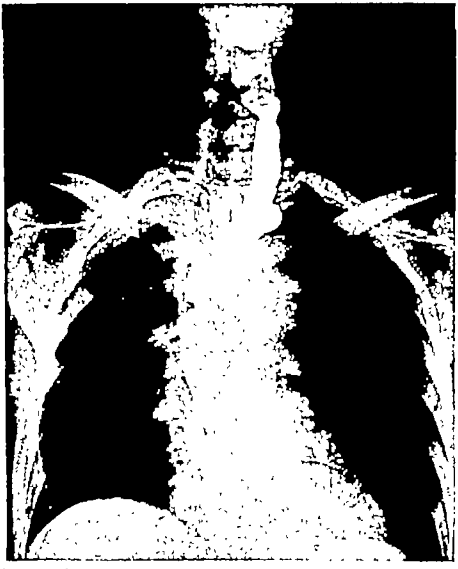

Fig. 8.—Barium swallow x-ray picture taken on the ninth postoperative day. The barium passed promptly into the distal esophagus through wide open stomas at both ends of the inert tube. The swallowing force was good and peristalsis of the distal esophageal segment was present and seemed to have an appreciable effect in emptying the replaced segment.

to administer x-ray therapy. A complete course of radiation was given to the neck through four portals, anterior and posterior and right and left lateral. The patient experienced a severe radiation reaction, and for a while it became difficult for her to swallow. With regression of the reaction, swallowing became better, and at the time of discharge from the hospital on April 30 the patient was again on a normal diet.

Comment.—It is obvious that this patient cannot be expected to be a long survival. The reconstructive procedure was carried to completion in spite of the demonstration of persistent carcinoma. However, at the present time the patient is well and without clinical evidence of recurrence. The operation was complicated by the presence of a large goiter in the neck; consequently, a bilateral neck dissection was not done. We feel that the neck dissection should be an integral part of the procedure, and the technique of this procedure will be the subject of a future presentation.

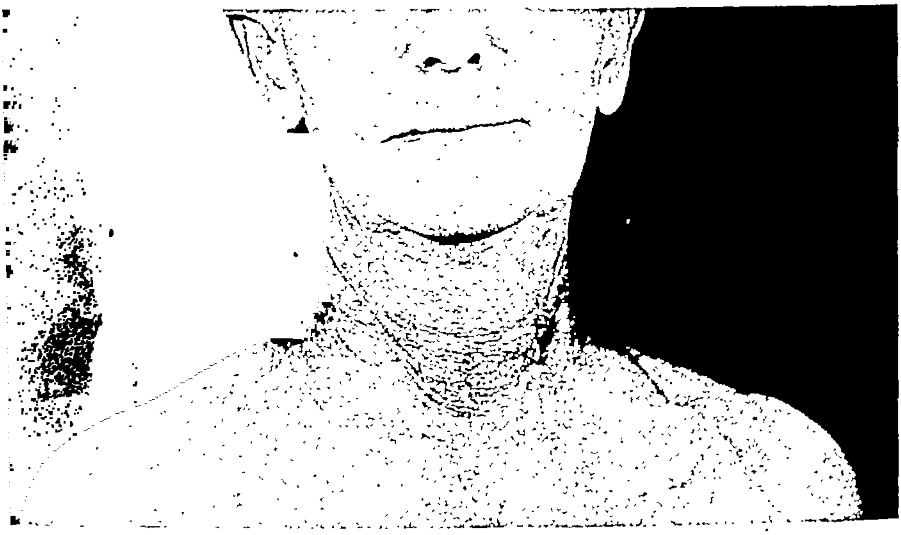

Fig. 9.—Photograph of patient nine months after completion of operation, showing normal appearance of neck and chest wall.

A point of particular interest to us has been the retention of active peristalsis in the distal segment. When a thick barium meal is given, the barium column goes rapidly and readily down to the lower anastomosis site where immediate active peristalsis begins with the result that almost instantaneously the reconstructed portion of the food passage is sucked empty. This, we believe, has very important implications.

DISCUSSION

While such a small experience is never too imposing, we feel justified in reporting it since certain important principles are involved. First, it appears that the rational approach to cancer of the upper third of the esophagus requires resection of the lymph nodes most likely to be involved. Second, a combined cervicothoracic resection and bilateral neck dissection is a feasible procedure, and, in our opinion, a less extensive assault falls short of the modern standards for cancer surgery. Moreover, this experience demonstrates the possibility, for the first time to our knowledge, of utilizing tubed pedicle grafts within the mediastinum. Of the greatest interest perhaps is the highly satisfactory functional result obtained.

We feel that with future cases the time can be greatly shortened so that the entire procedure can be completed within eight to ten weeks.

The applicability of this procedure to high localized strictures of nonneoplastic origin which are not suitable for esophagogastrostomy is, of course, obvious. In such cases the entire procedure can be completed much more quickly.

We wish to stress that in no sense are we unmindful of the magnificent advances stemming from the principles of one-stage resection with esophagogastrostomy. Our hope is that the procedure described here will prove to be a useful addition to the armamentarium of the surgeon in his struggle with cancer of the upper esophagus.

Grateful acknowledgment is made of the valuable advice and criticism we received from the members of the Plastic Surgery Service of Barnes Hospital.

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DISCUSSION

DR. FRANCIS O'DONNELL, Ann Arbor.—We have had an opportunity at the University of Michigan to see a case of the type mentioned by Dr. Burford. We, however, treated it differently. We performed a total esophagectomy, anastomosing the stomach to the pharynx. (Slides.) The patient was admitted in February, 1948, with a six-month history of progressive dysphasia. He had been reduced to a liquid diet for three months. He was in excellent general health. X-ray examination showed a stricture 4 cm. above the suprasternal notch. Esophagoscopy revealed the tumor to lie 3 cm. below the upper end of the esophagus. Biopsy showed a scirrhus carcinoma; there was no evidence of metastases.

The patient refused a flap operation and left the hospital, but returned in two months, having lost thirty pounds and having been completely obstructed for five days. Roentgenogram showed complete obstruction. The stomach was mobilized and brought to the apex of the chest. Of interest is the fact that the stomach was brought behind or to the right side of the arotic arch, since it is found that the esophagus takes the shortest and most direct route. A cervical incision was then made and the thoracic esophagus was brought out through the incision.

The incision on the tenth postoperative day consisted of a transverse incision and extension up the left sternomastoid. The cervical esophagus and the inferior 3 cm. of the pharynx were then freed and, drawing the stomach up into the neck, an anastomosis was made and the esophagus was removed.

(Slide.) This is the resected specimen showing the upper end—the free piece of tissue is the abdominal portion of the esophagus which was removed so that closure of the stomach could be through the gastric wall rather than the esophageal wall. When the anastomosis was performed at the neck there was free bleeding from the stomach, as a quarter size piece of gastric wall was excised preparatory to the anastomosis. Much to the relief of the surgeon, five ligatures had to be placed in the submucosal bleeding vessels of the gastric wall. The chest and neck were both drained. Penicillin was given for twenty days postoperatively and streptomycin for five days. The postoperative course was relatively benign except for a diarrhea on the seventh postoperative day, which might have been foreseen because of the extensive vagus resection. The diarrhea ceased on the thirteenth day.

(Slide.) This shows the thoracic and cervical stomach as seen on the twelfth day. There are two points of interest. On the right side can be seen the pylorus lying about 4 cm. below the diaphragm. The duodenal loop is somewhat straightened because of mobilization of the duodenum and moving it toward the left. At the left can be seen the stomach extending high into the neck, the barium just opposite the hyoid bone. We were unable to time our x-rays to catch the barium as it went through the pharynx and stomach. We had one severe problem postoperatively. There was no inferior constrictor muscle because it was excised. This led to small ulcerations about the mouth. They were treated by giving small amounts of alkali and the head of the bed was elevated to prevent regurgitation. He has gained twelve pounds since operation. Dr. Alexander sent me a wire last night saying the patient's temperature is normal and he is eating very well.

DR. RICHARD H. SWEET, Boston.—I was intensely interested in this presentation and congratulate Dr. Bricker and his associates on the successful accomplishment of something I have had in mind for a long time. I think one of the most significant things is that they lay emphasis in the correct place; we are dealing with a malignant tumor which has probably already metastasized to the regional lymph nodes when the patient comes in for treatment. In other words, any attempt at cure without taking out the regional lymph nodes will be doomed to failure. I have had a great deal of early enthusiasm for Dr. Wookey's procedure, but although I have had great success with function after the procedure—the ultimate result has been disappointing. All patients have died of recurrent or metastatic disease.

(Slides.) If we can relieve the patient so that he can swallow for the rest of his life that is probably all we will accomplish. In this case we were fortunate in catching some of the barium going through the anastomosis.

I think we should try the technique described here, but I am sure in my hands it would be doomed to failure, as was the Wookey procedure, unless we can take out the regional lymph nodes.

DR. JOHN R. PAINE, Buffalo.—I should like to tell of one patient operated upon with minor variations of Dr. Wookey's technique. This was a 53-year-old woman who had been obstructed for three months.

(Slides.) This is the x-ray film taken in 1947 showing the obstruction in the lower portion of the cervical esophagus. We used the Wookey technique, making a flap on the front of the neck. It was excised down to the upper border of the third dorsal vertebra. The larynx was involved in this case. Three biopsies had been reported as negative. At the time of operation the pathologist was willing to say it was a neoplasm, but did not say what the character was.

This picture was made three months later. It is hard to indicate exactly where the anastomosis is. The segment extends from the top of the third dorsal vertebra up to the pharynx. This is the late view after reconstruction of a skin tube. The barium will be seen in the sinus formed after the second operation. Four attempts were made at closure without success, and when the patient came back in one week they had closed of their own accord. After study of the section, several pathologists have told me it is some variety of sarcoma or endothelioma, but they are not definite as to type. Several lymph nodes were reported as negative after removal, and I hope this patient has some chance of cure.

This picture shows a trough through which she swallowed saliva. The second operation bridged this trough over.

DR. BRICKER.—I would like to thank the discussors, and particularly Dr. Sweet, for his encouragement. We are, of course, unable to argue strongly for this procedure on the basis of only one case. However, the enthusiasm which we admit is based on certain points which we think are sound:

First, regarding the cancer resection, we feel that a bilateral neck dissection and en bloc resection of the esophagus is the only procedure that can logically be expected to offer a reasonable chance of cure. Of course, if one decides that the disease is actually incurable, a view not too unjustified by survival figures so far, and that any surgery is simply palliative, aimed at allowing the patient to swallow comfortably for as long as possible, then the neck dissection can be discarded as a useless procedure. However, we disagree with this philosophy and feel that we should continue to aim at curability, and that time will prove that carcinoma of the upper third of the esophagus will not be controlled without regional lymph node removal.

The technique of bilateral neck dissection en bloc with the esophagus is a perfectly feasible procedure anatomically, which we hope to report in detail in the future.

Postericoid and higher lesions can be handled in a similar manner, the technical procedure being somewhat simplified by the sacrifice of the entire ribbon muscle compartment and larynx, with preservation of parathyroid function being the only problem. It is our opinion, admittedly not entirely crystallized at this point, that even the postcoracoid lesion should be handled as a combined cervicothoracic problem. This opinion is based on the necessity for wide resection of the esophagus itself, occasioned by the extensive submucosal lymphatic spread which we have witnessed and which has been noted by others.

Second, regarding restoration of the esophagus, our enthusiasm, which remains still to be justified, stems from the following points, most of which have already been covered.

1. The method provides a means of bridging a gap by choosing the shortest distance between two points.

2. The method provides the only means, to our knowledge, of preserving the propulsive force of the swallowing mechanism, plus the peristaltic force of the distal esophagus, plus a physiologic esophagogastric opening.

3. The method provides for intrathoracic reconstruction with tissue which has been prepared and for which the blood supply has been assured, rather than with tissue, such as jejunum or stomach, which must be carried in a one-stage procedure to the poorly defined limit of its blood supply and viability, and buried from sight.

4. The method does not require the disturbance or displacement of abdominal viscera in any way, except for the performance of a gastrostomy.

TRANSTHORACIC SMALL BOWEL SUBSTITUTION IN HIGH STRICTURE OF THE ESOPHAGUS

ALBERT WILSON HARRISON, M.D.
GALVESTON, TEXAS

THE construction of an artificial esophagus has challenged the ingenuity of surgeons throughout the world. That this challenge yet remains is testified by the multiplicity of approaches and methods used and by the perennial appearance of new techniques. A comprehensive summary of these techniques was published in 1944 by Yudin[3] of Moscow and his results in an extensive experience with 80 cases reflected the gratifying progress that had been made. Since then Longmire and Ravitch[1] have demonstrated the use of transplanted small bowel within a skin tube and even the preservation of its blood supply by anastomosis to the internal mammary artery. Rienhoff[2] has used small bowel transthoracically by reversing a length of jejunum back into the chest and anastomosing it antiperistaltically with the esophagus.

It is in the hope of contributing a bit further to the solution of this problem that the following cases are reported.

CASE REPORTS

CASE 1.—P. H. W. (Fig. 1), a 3-year-old colored boy, had had a Janeway gastrostomy almost two years previously for lye stricture of the esophagus. Investigation revealed the stricture to be practically complete at the level of the cricoid cartilage (Fig. 3) and all attempts at dilatation failed. It was obvious that if he were to eat again, some form of substitution would be necessary. To this end three stages of operation were planned. In the first, through an abdominal incision, a segment of jejunum was prepared with one arcade of blood supply and isolated. Jejunal continuity was re-established by end-to-end anastomosis. The isolated segment was brought up through the transverse mesocolon, its distal end being anastomosed to the fundus of the stomach and its proximal end closed. Two weeks later the left side of the chest and diaphragm were opened. The esophagus was dissected from the mediastinum and transected at the cardia. It was then brought out through an incision in the neck along with the proximal end of the jejunal loop. Excess esophagus was left for a few days to conduct saliva away from the neck wound.

After this stage an unplanned gastroenterostomy became necessary to relieve an obstruction at the pylorus. Apparently this was due to the combined effects of vagotomy and bow-stringing of the artery to the displaced segment across the lower stomach. At the third stage, two weeks following the second, the stricture was excised and the pharynx was opened to a diameter of 1.5 cm. by a posterior median slit. The jejunal segment was opened and a two layer end-to-end anastomosis was made to the pharynx.

A good result was obtained at first. The chest was clear (Fig. 5) and a barium swallow (Fig. 7) passed rapidly into the more tortuous bowel within the chest. Rapid and vigorous peristalsis delivered it into the stomach in less than one minute. Within a few weeks, however, swallowing became more difficult and an attempt at dilatation resulted in a pneumothorax. Four months after the third stage the pharyngojejunal anastomosis was excised and reconstructed with a much larger aperture. This time the child was discharged home, eating normally. He still swallows well four months later.

From the Department of Surgery, University of Texas, Medical Branch.

Read at the Twenty-eighth Annual Meeting of The American Association for Thoracic Surgery, Quebec, Canada, May 31, June 1, 2, 1948.

CASE 2.—C. B. (Fig. 2), also a 3-year-old colored boy, had had a Janeway gastrostomy after drinking lye at about the age of 16 months. Barium study showed a complete stricture of the esophagus just above the manubrium (Fig. 4). Operation was done in three stages as in the first case, but with certain notable differences. In the first stage the isolated loop was brought up behind the stomach and out through the gastrohepatic omentum, and in the second stage the vagus nerves were carefully preserved. The esophagus itself was unusual in that it had become obliterated and reduced to a fibrous cord in the thoracic inlet. Finally during the dissection it pulled out entirely. An artificial opening was made up through the cervical mediastinum and the end of the loop was implanted alongside the upper esophageal segment. In the third stage the upper esophageal pouch was found to be quite large and filled with wads of chewing gum, food, and other matter. To obtain a sufficiently wide anastomosis here was no problem.

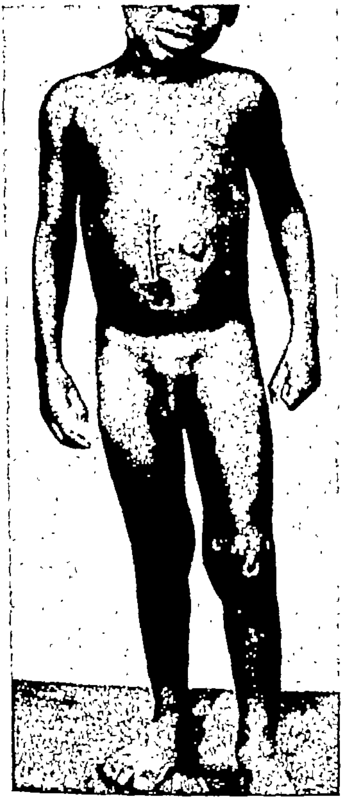

Fig. 1.

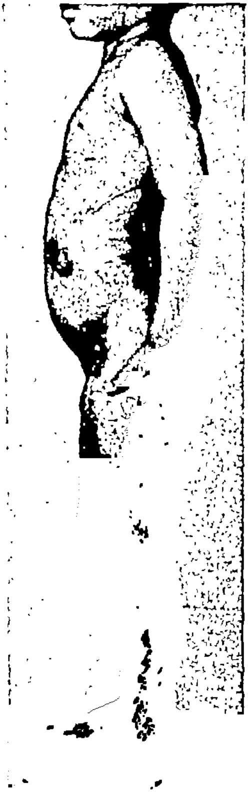

Fig. 2.

Fig. 1 (Case 1).—Photograph showing gastrostomy and scars of the various stages of operation.

Fig. 2 (Case 2).—Photograph showing lateral view.

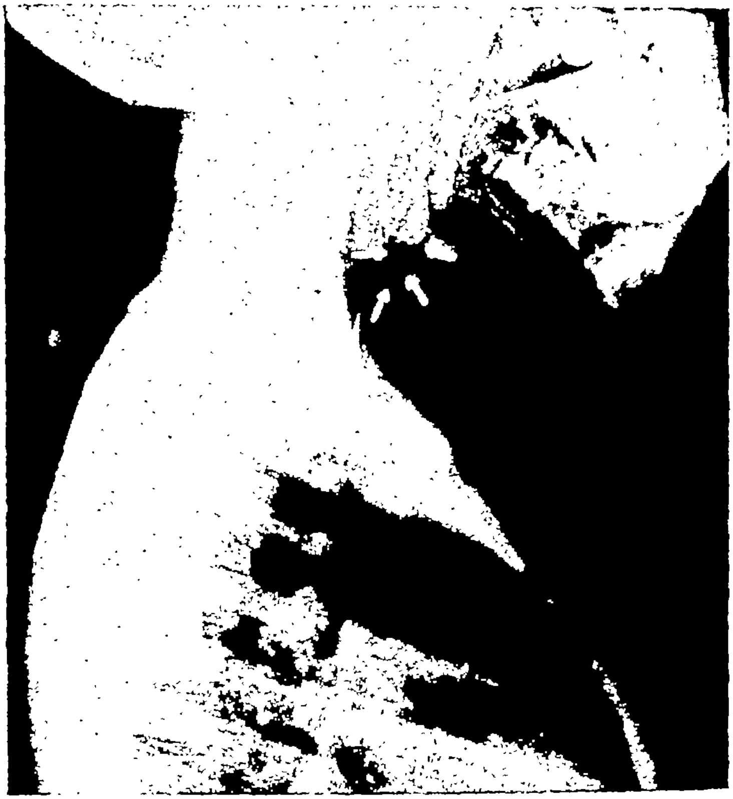

Fig. 3 (Case 1).—Arrows indicate outline of barium above the stricture.

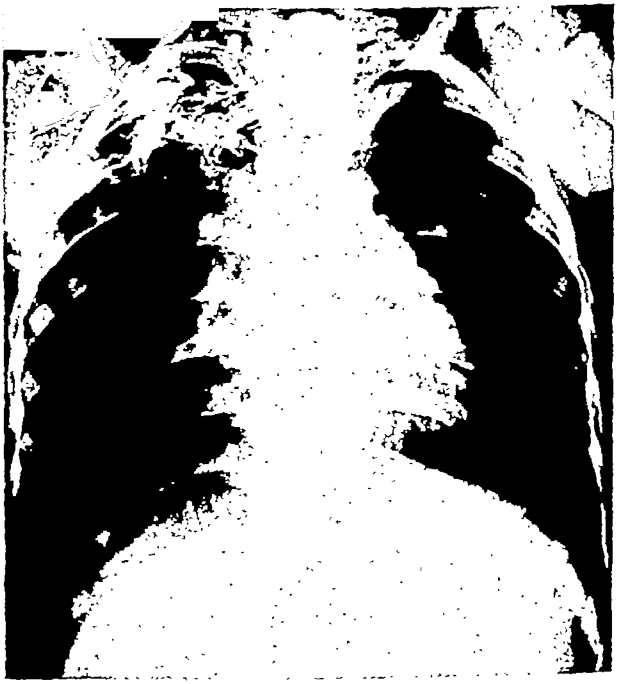

Fig. 4 (Case 2).—Dilated esophageal pouch filled with barium; extraneous barium on skin appears on the right.

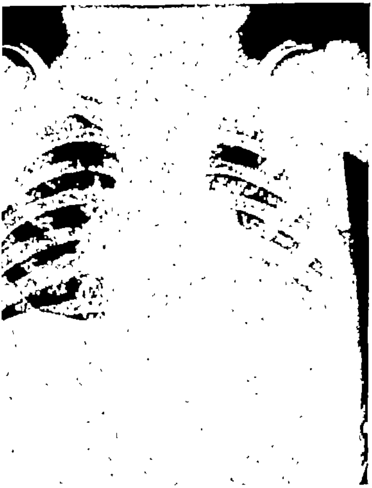

Fig. 5.

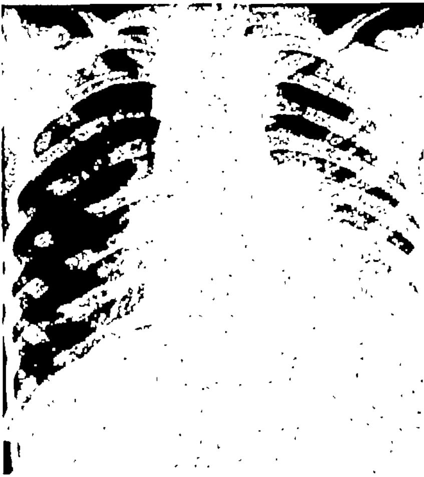

Fig. 6.

Fig. 5 (Case 1).—Postoperative chest film of patient showing dilated stomach.

Fig. 6 (Case 2).—Postoperative chest film of patient, relatively clear.

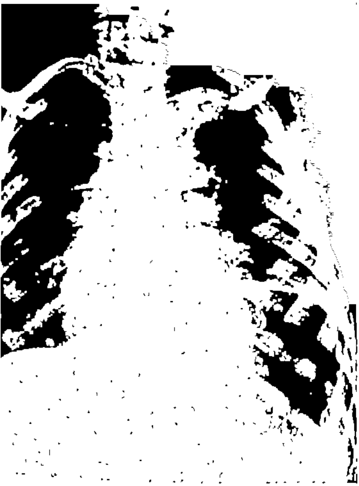

Fig. 7.

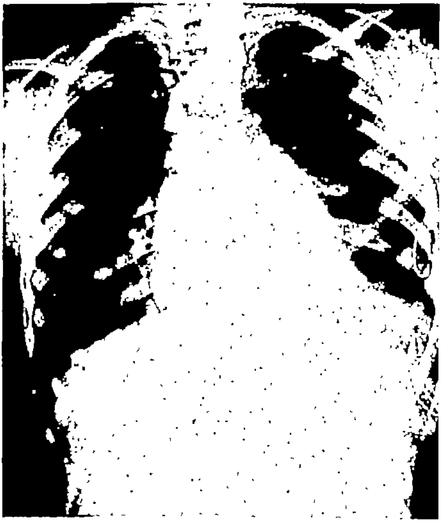

Fig. 8.

Fig. 7 (Case 1).—Barium swallow; the bowel is quite tortuous but peristalsis is vigorous and delivers barium into stomach in one minute.

Fig. 8 (Case 2).—Barium swallow, showing straighter bowel and more rapid progress of barium.

Postoperatively the chest was clear (Fig. 6) and the barium swallow (Figs. 8 and 9) dropped more or less straight to the lower thorax where peristalsis in the lower coils rapidly passed it on into the stomach. The patient was discharged in one month after operation, eating normally. Plans were to leave the gastrostomy open for a test period of one year or so before closure.

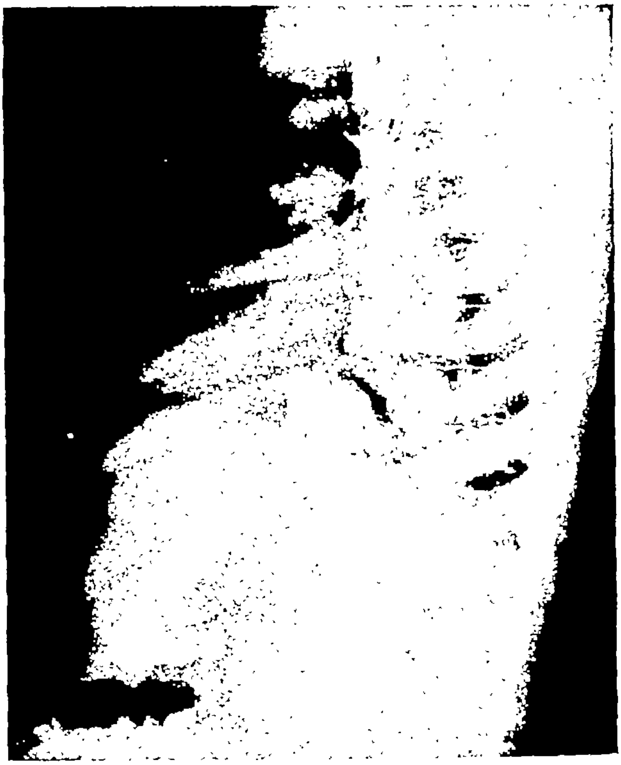

Fig. 9 (Case 2).—Lateral view showing bowel fairly straight in upper mediastinum, coiled in lower.

From these histories it is evident that three principal mistakes were made in the first case. (1) The antegastric transposition of the jejunal segment resulted in obstruction in the antral region of the stomach. (2) Vagotomy at the time the esophagus was resected rendered the stomach atonic and contributed to the obstruction. (3) The anastomosis at the pharyngeal junction was made too small because of fear of interfering with the mechanics of swallowing, whereas subsequent observation showed this danger to be more theoretic than real. There were no significant complications other than the ones mentioned. The thoracic duct fortunately was not encountered in either case and the recurrent laryngeal nerves were carefully avoided. The distal esophagus was resected both times to eliminate a potential source of danger in the mediastinum. All wounds healed per primam.

TECHNIQUE

The problem in the first stage of such an operation is to isolate a segment of jejunum whose blood supply will have adequate length to reach the neck without undue tension and without too much redundancy of bowel. To accomplish this a long arterial arcade is established by sacrificing as many intermediate radicals as seems necessary (Fig. 10), usually no more than one or two main jejunal branches. As shown in the drawings, the bowel is transected proximally where the secondary radicals are longest so as to give maximal upward offset of bowel along blood supply, thereby adding length to the proximal end. The distal end on the other hand should be cut as short as blood supply to the remaining jejunum will permit. This serves to minimize redundancy in the isolated segment, which is always a problem. The proximal arterial supply is not divided as in the drawing until the second stage.

The loop is brought up through the transverse mesocolon, the lesser peritoneal sac, and the gastrohepatic omentum where it is fixed to preserve maximum length above this structure. The proximal arterial radical is tagged for ready identification later. The continuity of the remaining jejunum is re-established by end-to-end anastomosis.

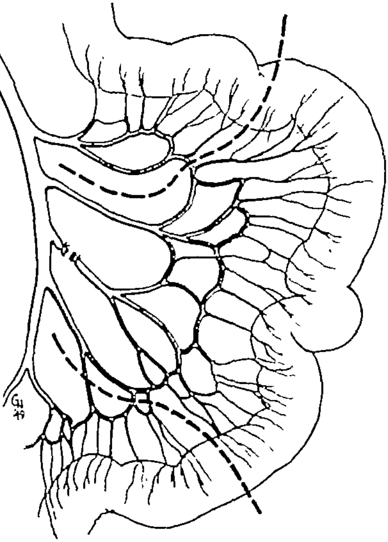

Fig. 10.

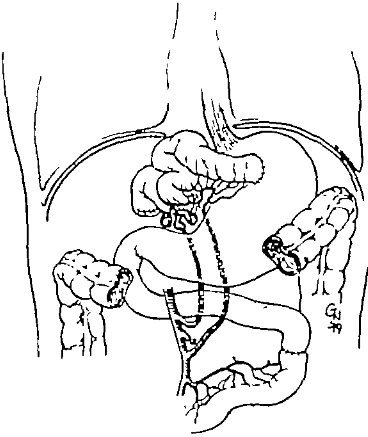

Fig. 11.

Fig. 10.—Plan for first stage of operation showing how bowel will offset upward on blood supply.

Fig. 11.—First stage completed. Isolated segment is actually coiled and covered with veil of *omentum to prevent adhesions. Proximal arm of arterial arch is not divided until* the second stage.

The distal end of the jejunal loop is anastomosed to a fairly large circular ostium in the fundus of the stomach made by excising a coinlike portion of its wall. The proximal end of the loop is closed. The loop is then coiled up, covered with a veil of greater omentum to prevent adhesions, and left beneath the diaphragm (Fig. 11).

In the second stage the chest is opened through the seventh interspace and the diaphragm opened anterolaterally for a short distance from the esophageal hiatus. The segment of jejunum is freed with extreme care to avoid tearing away seromuscular layer. The proximal arm of the arterial arch which was tagged previously is divided and the mesentery dissected along the remaining vascular supply to remove peritoneum, lymph nodes, and other tissues restricting length. Care must be taken not to place tension on the vessels which might injure them. The vagus nerves are mobilized from the esophagus and preserved. The esophagus is freed from the mediastinum and transected at the cardia. The opening in the stomach is closed securely to permit early feeding.

An incision is made along the anterior border of the left sternomastoid and both the esophagus and the upper extremity of the jejunal loop are drawn out the neck with as little tension as possible. The jejunal segment may pass on whichever side of the aortic arch that seems best. The loop inside the chest is straightened as well as possible and the excess is replaced in the abdomen where it is fixed as the diaphragm is closed. Penicillin is left

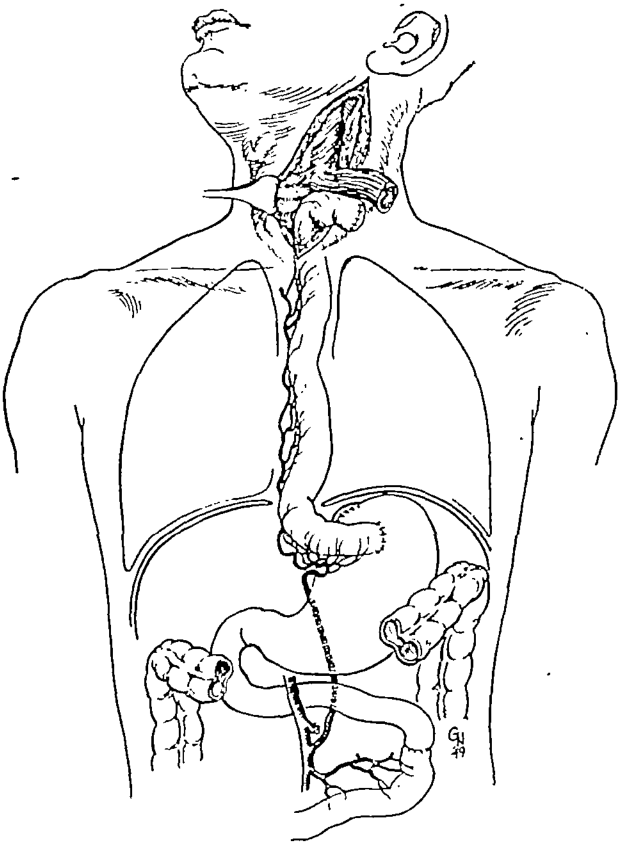

Fig. 12.—Second stage completed. Segment of bowel is much more tortuous than shown.

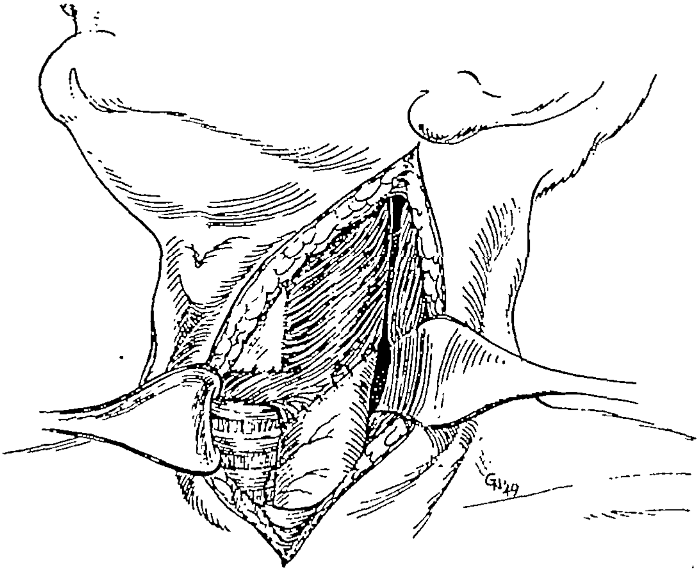

Fig. 13.—Anastomosis of esophagus to jejunal segment is complete.

in the pleural space and two or more intercostal drainage catheters are employed for a few days to insure early and complete re-expansion of the lung. The resulting picture appears in the drawing (Fig. 12).

After the mediastinal space is well sealed, the anastomosis can be made in the neck without fear of possible leakage (Fig. 13). The anastomotic aperture should be made as large as possible by beveling or some such device. A small rubber tissue drain should probably be left in the retroesophageal space postoperatively.

DISCUSSION

Although two cases represent a very limited experience, certain essential features have been observed to contribute most directly to the outcome. (1) In the first stage the jejunal loop must be carefully planned to obtain maximum length of blood supply and minimum length of bowel on its distal end. Although it has not been tried, it would seem feasible even to sacrifice some of the redundant portion of bowel to get a "straight shot" to the stomach. (2) The loop must be transplanted behind both colon and stomach to take full advantage of the length of the artery and to avoid obstruction to stomach or bowel. (3) The loop is closed at its proximal end and anastomosed to stomach at its distal end at the first stage to avoid contamination of the mediastinum during the second stage. (4) A fair-sized coinlike portion of the stomach should be excised for anastomosis to insure adequate centrifugal pull by gastric muscularis for maintaining patency. (5) Extreme gentleness and care are necessary at all times if adhesions are to be avoided. (6) The vagus nerves should be preserved if at all possible. (7) The blood supply should be carefully liberated to its fullest extent by dissecting away peritoneum and lymphatic tissue. (8) Closed drainage of the pleural space postoperatively hastens closure of the mediastinum and prevents infections. (9) The third stage is delayed until the mediastinum is well sealed so there will be no danger should the cervical anastomosis leak. (10) And finally the anastomosis in the neck should be as large as feasible.

The advantages of this method seem to be as follows: (1) The blood supply to the transplanted loop is routed through the shortest most direct path to the neck, thereby creating less difficulty in obtaining adequate circulation. (2) The substitute segment of bowel is shorter and straighter and the ingested bolus "drops" more rapidly to the stomach. This avoids the relative stasis of longer segments. (3) The loop is isoperistaltic and responds well to the stimulus of distention. (4) The stomach retains its normal function regarding both capacity and digestion. (5) Two potential danger points, the anastomosis sites, are outside the chest and thus more accessible if difficulty should arise. (6) The gastrostomy is not disturbed throughout the entire procedure so that there is always a satisfactory means of maintaining nutrition. (7) Last but not least the cosmetic effect is much more satisfactory.

SUMMARY AND CONCLUSIONS

Two case reports have been presented in which complete lye strictures of the cervical esophagus were treated by transthoracic substitution of a loop of jejunum for the esophagus. The three stages of operation for this have been described and the essential features pointed out. The advantages have been enumerated, the disadvantages being taken into account. So far the results have been very encouraging and further trial seems indicated.*

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DISCUSSION

DR. HERBERT WILLY MEYER, New York.—This interesting paper of Dr. Harrison's is an important contribution to the surgery and reconstruction of the esophagus. The intrathoracic use of a single tube of jejunum to replace the esophagus is a real advance. In 1940 we had to resect the lower thoracic esophagus together with a total gastrectomy for carcinoma. An intrathoracic esophagojejunostomy was employed to re-establish continuity of the intestinal tract. A double-barrel loop of jejunum was brought up into the thorax with an enteroenterostomy lower down within the abdomen. The Roscoe Graham type of anastomosis was employed. There was definite tension at the suture line. We briefly reported this case at the Toronto Meeting of our Association and the case was published in *Surgery* in 1942. I should like to present one lantern slide to show the functioning anastomosis four months after the operation. Dr. Churchill at that time stated that he had performed a similar operation but the patient unfortunately did not survive. We both had found that there was a good deal of tension at the site of the anastomosis if a double-barrel loop of jejunum was employed.

Dr. Harrison's method of using only a single tube of jejunum is a real advance and completely overcomes the objection of tension at the anastomosis. I am certain that this newer and better method will be employed frequently in performing intrathoracic esophagojejunostomy.

DR. RICHARD H. SWEET, Boston.—I hesitate to address you again, especially after such a brilliant procedure which is far better than any of my own experiences in this field, but I would like to tell you that I have also been trying to "out-Rienhoff Rienhoff."

(Slides.) This was another example of the use of the jejunum in the mediastinum, and I am absolutely convinced that that is the proper place for it. I think it is far superior to any external esophagoplasty of any sort whatever, especially using the jejunum. This was a case of lye stricture of the esophagus involving the entire esophagus from the pharynx down.

This is the resected specimen and, because of my preference for one-stage operations, I performed this in one stage with two phases, the abdominothoracic as the first incision, freeing a loop of jejunum, bringing it up and then after a sufficient quantity of jejunum had been obtained, closing the chest incision and making the cervical one. This shows the swallow of barium going down through. The operation is not an easy thing to accomplish, and I am sure Dr. Harrison's method has certain superiorities. This loop was brought up and after transection the proximal end was implanted low down into the distal portion by the Roux type of procedure. The other end was used for the pharyngojejunal anastomosis.

The reason I did not use the stomach in this patient was that she had had the antrum resected because this lye had not only burned the entire esophagus but had also produced a stricture at the pylorus. She had had a partial pylorectomy which made it impossible to mobilize the stomach.

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*At the time of this publication, one year later, both patients eat well and are apparently without complication or difficulty.

DR. WILLIAM F. RIENHOFF, Baltimore.—We have had three successful cases of intrathoracic transplantation of the jejunum following resection of the esophagus and I intend to use this method more frequently in the future, for the simple reason that the jejunum, from the standpoint of length available, prevents difficulties and the size of the eventual tube is better suited to take the place of the esophagus in the mediastinum than pulling up the bulky stomach. I was very much interested in Dr. Harrison's presentation and Dr. Sweet's discussion. Dr. Harrison's leaving the first branch of the vasa recta jejunalis may have a very important role in preventing scarring and curling of the jejunum which is left in the abdomen following mobilization. Dr. Blalock, some years ago, suggested to me that it would perhaps be better to leave the jejunum in the abdomen for a few days after mobilization by cutting the first, second, and third recta jejunalis, dividing the proximal end of the jejunum just distal to the ligament of Treitz. However, it was found that when this was done the jejunum became curled and the vascular comb was shrunk and scarred to the extent that it was very difficult to affect a thoracic bridge or spread out the jejunum sufficiently to bring it into the chest cavity. Attempts to straighten the jejunum would often tear the mesentery and result in interference with circulation to the bowel. These patients were reported in the *Southern Medical Journal* in December, 1946.

A reversed Roux loop, leaving the first vessel departing to the jejunum from the superior mesenteric artery, was performed by me and left in at this time. This left the first artery intact, as suggested by Dr. Harrison. Perhaps some form of transplantation of vessels into the mesentery of the jejunum intrathoracically might be the answer to the problem.

The difficulty in using jejunum heretofore has certainly been one of interference with circulation to the bowel. In a patient reported by Dr. William Longmire, in which he did a subcutaneous transplantation of the jejunum, the internal mammary artery was sutured to one of the jejunal vessels of the vascular comb. This method, however, on account of the very small diameter of the vessel would probably not be practical in the majority of cases and, furthermore, such a vessel could not be used intrathoracically because not enough length could be obtained. If the circulation of the jejunum is maintained, and I think it can be, there would seem to be no question of a doubt as to the desirability of the use of jejunum in preference to the stomach.

DR. CLARENCE CRAFOORD, Stockholm.—I do not know if information is available in this coutry about the work that Yudin in Moscow has done in this field. I have not been able to obtain any accurate information myself, but from Russian surgeons who have visited Stockholm I have heard that he has now done between 200 and 300 operations of this type, anastomosing the jejunum with the esophagus as high up in the neck as the pharynx. In this number are included, I have been told, both cases done in many stages and one-stage procedures, both in front of the sternum and inside the chest. He has also tried the same method that Dr. Harrison has described, but he has found that later on in these stumps, transplanted into the pharynx, there has developed severe irritation even with ulcerations, so he now strongly advises not to anastomose the jejunum with the stomach. He advises letting the food go directly from the mouth into the jejunum, excluding the stomach.

I have performed only one of these operations, and it was astonishing to me to see how long a loop it was possible to obtain with very good circulation by dividing the vessels after the method shown by Dr. Harrison. One can divide four or five branches and get a loop that is about 75 to 100 cm. long, which carries up into the neck without difficulty.

DR. OWEN H. WANGENSTEEN, Minneapolis.—Dr. Harrison's report has been very stimulating. There is an additional manouver that may be implemented to insure a better arterial blood supply to the jejunal loop swung up into the thorax, a procedure which I have employed several times as an auxiliary to total gastrectomy, with the thought in mind that the capacity of the new jejunal reservoir for food would be increased thereby. The technique consists in denuding the superior mesenteric artery at its root from the aorta from the overlying adventitious tissue; in other words, the superior mesenteric ganglion is removed. This procedure should not only delete the anastomotic arterial channels of the small intestine but should, at the same time, diminish the resistance to distention of the loop, thereby, theoretically at least, making it possible for the patient to eat larger meals without experiencing intestinal colic.

I have had no experience with the procedure described by Dr. Harrison of bringing segments of jejunum up within the thorax. Whether one should anastomose the distal end of that segment to the fundus of the stomach as Dr. Harrison did or re-establish continuity by an end-to-side jejunojejunostomy, as Yudin does and Crafoord prefers, is difficult to decide. An isolated gastric pouch into which neither food nor saliva make their way may invite a duodenal ulcer; on the other hand, may a jejunal ulcer develop at the new jejunogastric anastomotic site if the new esophagus (jejunal segment) is anastomosed to the stomach? In other words, both procedures leave questions unanswered, to be resolved only by time and more experience. In defense of the operation as Harrison did it, there is at least this consideration: a jejunogastric inlet anastomosis probably does not invite a stomal ulcer in the same degree as a gastrojejunostomy outlet, because in the latter instance the stoma is an important outlet for the acid-peptic digestive juice, whereas, in the instance of the jejunogastric inlet anastomosis, there will be only occasionally regurgitation of gastric juice into the proximal jejunal segment. Whereas Dr. Harrison believes it best to leave the vagi nerves in order to avoid gastric retention, vagotomy by reducing gastric secretion may diminish the hazard of a stomach ulcer. If a vagotomy is done, a pylorotomy of the Rammstedt type probably should be added.

There is an additional comment which I should like to make with reference to the nature of so-called idiopathic esophagitis and stricture. The experience of our surgical clinic with the management of these lesions suggests that esophagitis is a manifestation of ulcer disease. My associate, Dr. N. Logan Leven, and I have now observed six patients in whom gastric resection did away with the necessity for continued dilation of an esophageal stricture. The first patient was observed in 1939; an extensive gastric resection was done because of massive hemorrhage for a duodenal ulcer. During the previous five years, the patient's esophagus had been dilated more than 100 times. After a few dilations following gastric resection, the necessity for dilating the esophagus disappeared. During the intervening years, five additional patients with esophageal stricture have been treated similarly with equal success. Some of these patients have had an accompanying ulcer, others have not; one patient had a corkscrew-like stricture of the lower esophagus associated with a congenitally short esophagus. I am now considering the idea of exploring the possibility of whether the dysphagia of cardiospasm as well as that of bona fide lye stricture (of the lower esophagus) also may respond to gastric resection, on the thesis that an active acid-peptic secretion may continue an established vicious circle.

Moreover this much also probably should be said: the esophagus undoubtedly is more susceptible to injury by the acid-peptic digestive juices than is the stomach or the duodenum. And if the esophagus rather than the duodenum were the gastric outlet, I believe that every patient with an active gastric secretory mechanism would have an acid-peptic ulcer in the esophagus. Work now in progress in our experimental laboratory shows definitely the greated vulnerability of the esophagus to injury by the gastric secretions. If a dog's duodenum is obstructed just beyond the pyloric outlet and histamine is given to stimulate gastric secretion, esophagitis with perforation is a common sequel, whereas the stomach in the short postoperative survival period appears to escape unscathed.

DR. HARRISON.—It is indeed gratifying to hear the encouraging comments of these distinguished gentlemen. In answer to Dr. Rienhoff's question, certainly the bowel is inclined to shorten and become adherent, and the mesentery contracts. The purpose in very carefully wrapping this loop of bowel in available omentum is to prevent these adhesions. It seemed to me in these two cases that by carefully dissecting the mesentery and teasing away the restricting tissues the loop should gradually straighten out to the necessary length.

COMPLETING THE MULTIPLE-STAGE OPERATION FOR ATRESIA OF THE ESOPHAGUS

CONRAD R. LAM, M.D.
DETROIT, MICH.

IN 1943, Haight and Towsley[1] reported the first successful surgical repair of congenital atresia of the esophagus by a single mediastinal operation. Since then, there has been general agreement that this plan of attack is far superior to the multiple-stage procedure which resulted in a fair number of surviving children, most of whom, however, carried salivary fistulas and gastrostomies for a long time while they waited for the final construction of the antethoracic esophagus. The improved prognosis in infants treated by primary end-to-end anastomosis is illustrated in Swenson's[2] excellent series. He was able to complete the operation in thirty-two out of thirty-seven cases, and, of these, twenty-seven survived.

In my series of thirteen infants, it was possible to perform a presumably satisfactory anastomosis nine times. Only three of these babies survived; the unsatisfactory results were due to breakdown of the anastomosis four times and anesthetic difficulties twice. Of the four babies who did not have an end-to-end anastomosis, two died in the immediate postoperative period following intramediastinal ligation of the fistula, and futile attempts to connect the segments of the esophagus. The other two children have had the continuity of the alimentary tract established by the construction of an antethoracic esophagus when they were relatively young (at ages 27 months and 18 months). It is with the surgical problem of completing such multiple-stage procedures that this paper is concerned.

As will be seen from the case reports, the sequence of events differed considerably in the two infants. Nevertheless, the final anatomic situations were identical, and the following general plan of management was evolved. If approximation of the two segments is impossible at the time of the mediastinal exploration, the lower segment is detached from the trachea, ligated, and allowed to retract toward the abdomen, and the tracheal opening is closed (Stage 1, Fig. 1). Two days later, if the condition of the infant warrants, the upper segment is exteriorized in the left clavicular region and is opened to permit sham swallowing (Stage 2, Fig. 1). The third stage follows shortly; it consists of mobilization of the cardiac end of the stomach and the lower esophageal segment, through an abdominal approach. The segment is exteriorized through a skin tunnel, the end being brought out as high as possible on the chest wall (Stage 3, Fig. 1). Feedings through this esophagostomy are carried out during the ensuing year by the intermittent insertion of a catheter. Such an esophagostomy is better than a gastrostomy, because the intact cardiac sphincter retains the stomach contents after the removal of the tube and there is no excoriation of the

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skin. Carter[3] suggested such use of the lower segment, but in his one case, an abdominal operation only was done and only that part of the esophagus which could be drawn down through the esophageal hiatus was available. The infant lived only a few weeks. It may be significant that there is no record of an ultimate survival when a blind esophageal pouch has been left attached to the trachea.

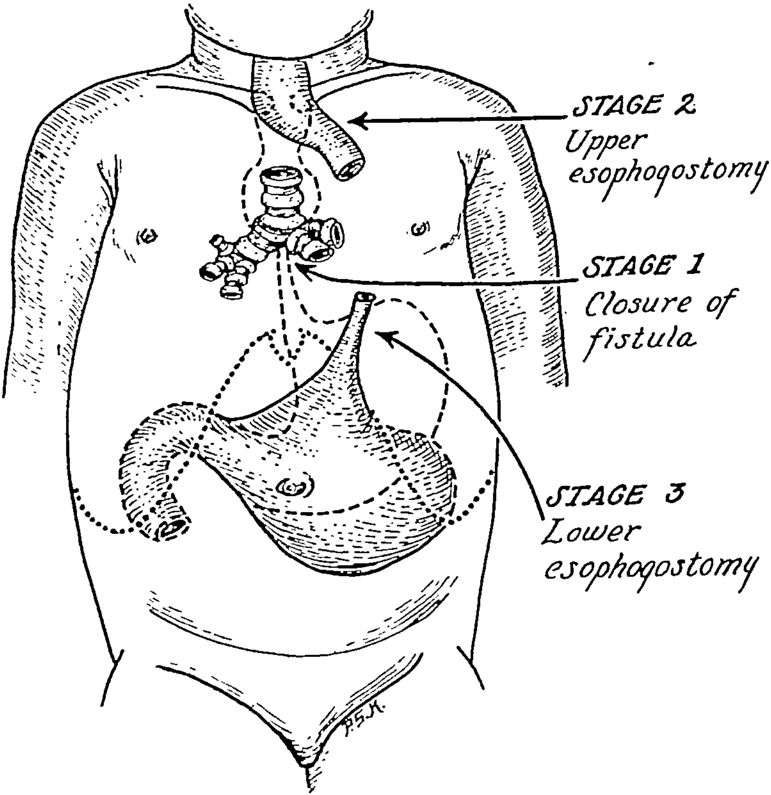

Fig. 1.—Plan of first three operations which may be carried out in the first week if a multiple-stage method of management of congenital atresia of the esophagus is necessary.

During the second year of life, the two esophagostomies are connected by a skin-lined tube. The plan of this phase is illustrated in Fig. 2. Theoretically, the tube could be constructed in three stages, (1) preliminary formation of the tubed pedicles (not shown in Step A, Fig. 2); (2) formation of the tube proper, with covering of its raw external surface with adjacent skin and/or the tubed pedicles (Steps A to D, Fig. 2), and (3) final severance of the pedicles (Step E, Fig. 2). Actually, in the two cases reported below, a considerably larger number of operative procedures were required. Most of the difficulties resulted from attempted short-cuts to avoid the use of tubed pedicles, and from strictures at the esophago-dermic junctions.

Case 1.—T. R. A preliminary and a progress report on this child have been made in previous communications.[4, 5] He was born in the Henry Ford Hospital on Aug. 1, 1943, and the diagnosis of atresia with tracheo-esophageal fistula was made when he was three days old. Because of the high mortality then associated with the mediastinal operation, I elected to do the multiple-stage procedure, although I have no doubt that the direct

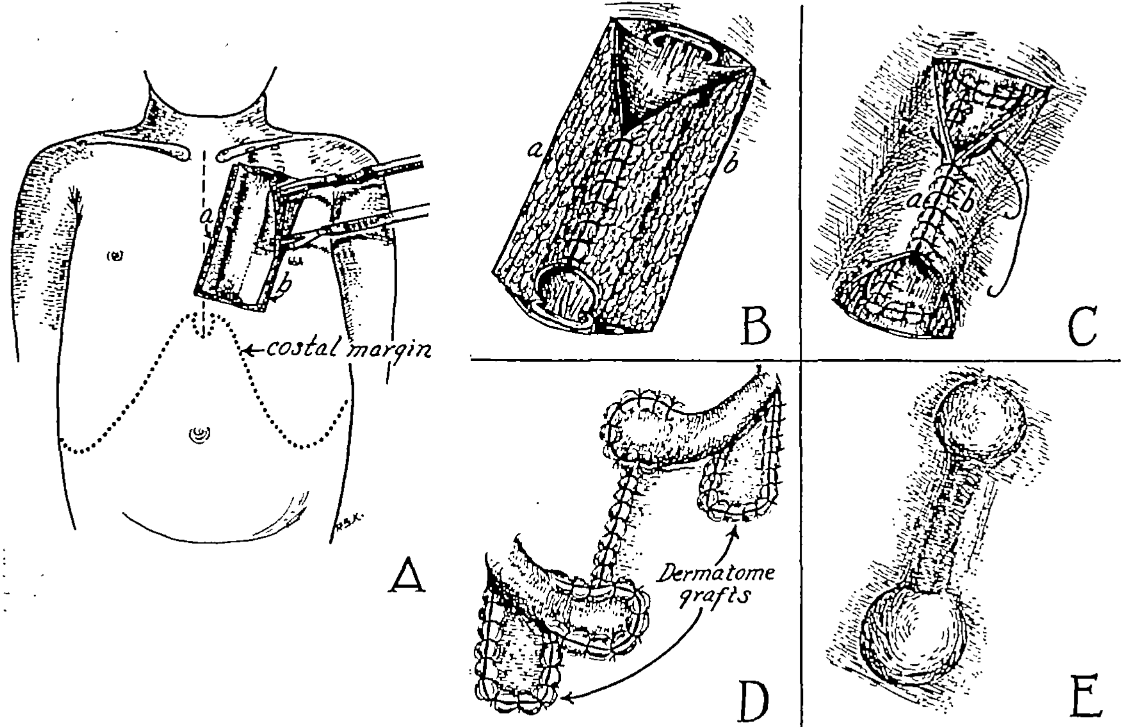

Fig. 2.—Steps in the formation of a dermic tube to connect the two esophagostomies. This stage may be begun when the child is 1 year of age.

operation would have been anatomically feasible. On August 4, under drop-ether anesthesia, the stomach was transected as near the cardiac orifice as possible and mushroom catheters were placed in the two parts of the stomach. The one on the pyloric side was used for feeding and the one on the cardiac side was used to drain the remaining lower segment of esophagus. Such a procedure was once used by Gamble,[6] whose patient died on the twelfth day from perforation of the stomach. It was similar in principle to a plan followed by Leven[7] on a child who is now the oldest patient to survive any kind of attack on the anomaly.

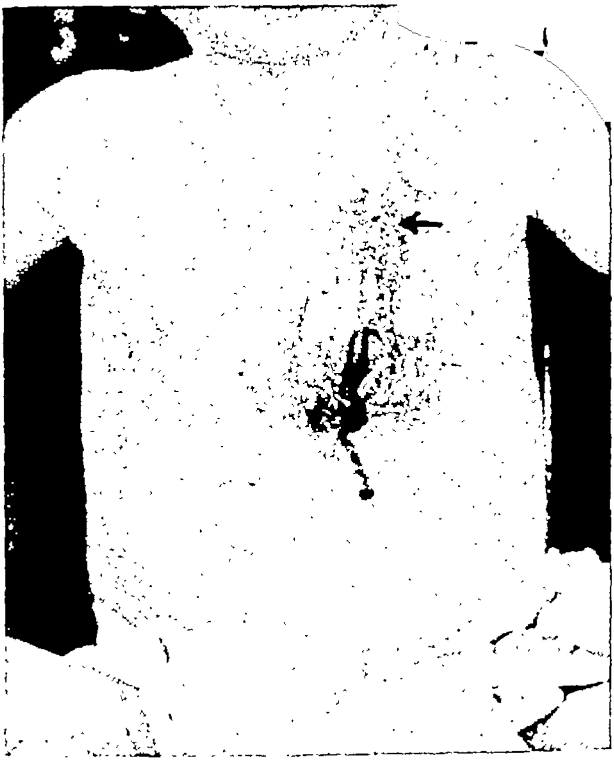

Fig. 3 (Case 1).—Photograph taken after the first attempt to form the skin tube. Note the upper small defect (arrow) and the larger lower defect which has been temporarily bridged with a piece of rubber tubing.

On August 18, the upper segment of esophagus was exposed through an incision along the anterior border of the left sternomastoid muscle, and its length was such that the end could be exteriorized through an opening in the skin below the clavicle. (Stage 2, Fig. 1.) At the age of one month, he was discharged to home care. The plan was to allow him to attain the age of one year before anything further was done. However, on Jan. 21, 1944, he was admitted as an emergency case because he had coughed up some food which had been placed in the distal gastrostomy. It was obvious that a fistula had formed between the two portions of the stomach, and it was urgent to disconnect the lower segment from its fistulous connection with the trachea. On January 24, I opened the left pleural cavity through the seventh interspace. The esophagus, which was the size of the little finger, was picked up and traced to where it joined the trachea at its bifurcation. It was doubly ligated at this point and cut. The diaphragm was opened and the cardia was mobilized by cutting the short gastric and left gastric vessels and the end of the esophagus was brought out through a stab wound in the left lateral abdominal wall. It could not be brought to its final location on the anterior chest wall because the two gastrostomies were in the way. On February 11, the continuity of the stomach was re-established and the two gastrostomies closed. Feeding was now easily accomplished by the intermittent insertion of a catheter into the lateral esophagostomy. On February 19, he was discharged again for home care.

On Aug. 10, 1944, when he was a little over 1 year old, he was admitted for the construction of the antethoracic esophagus. As a first step, the lower segment of esophagus was moved from its position on the lateral abdominal wall to a point above and to the left of the xiphoid. Thus, the situation in Stage 3, Fig. 1 was not accomplished in this patient until he was a year old. On September 8, an attempt was made to construct a skin tube connecting the two esophagostomies according to steps A, B, and C in Fig. 2. Due in part to malnutrition, the skin of the chest wall was so loose that I yielded to the temptation to close the triangular defects seen at each end of the tube in Step C by continuing the linear suturing until the entire raw surface of the tube was covered without the use of free or pedicle grafts. The result looked very good immediately after the operation, but on the fourth day, there was opening up of the tube at the lower end for a distance of several centimeters. The main factor in this appeared to be adherence of the two suture lines, with the result that pull on the external skin also tended to pull the suture line of the dermic tube apart. For this reason, it has been indicated on Step C, Fig. 2, that if such a closure is contemplated, the suture line in the dermic tube should be eccentrically placed so that it will not lie immediately under the outside suture line.

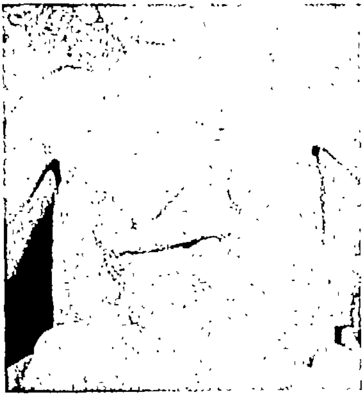

Fig. 4 (Case 1).—A tubed pedicle is being utilized to cover the lower defect.

The appearance of the abdominal wall after this stage is shown in Fig. 3. In addition to the large lower defect, there was a small leak higher up. Further attempts to complete the tube were postponed until the tissues were softer. Then on February 5, the lower part of the tube was closed again, and the raw surface was covered with a dermatome graft. This stage was also a failure, and it became obvious that nothing short of a substantial pedicle graft would give permanent closure. Consequently, on February 19, a pedicle was raised on the right side of the abdomen, and on March 5, this was used to cover the lower part of the dermic tube which was formed for the third time (Fig. 4). On April 30, the pedicle was cut loose from the right side of the abdomen and the excess tube was attached at a point higher on the chest in order that the part not needed for the lower defect could be used for a patch over the upper defect. This procedure was done on May 18. The upper opening did not remain closed, and it was utilized as an opening through which to dilate both the upper and lower esophagodermic junctions with olive-tipped dilators (up to size 8 common duct dilators). The stricture at the lower junction was so tight that on September 7, its posterior aspect was exposed and it was corrected by a plastic procedure, a longitudinal incision being closed transversely. Dilatations were continued by passing the dilators through the upper opening until it appeared that a satisfactory lumen would be maintained. On Nov. 15, 1945, the edges of the upper opening were freshened and it was closed by suture. This was the last of twenty trips to the operating room. There has been no leakage since, and the child has consumed a full diet at the family table without interruption. Fluids and soft food are ingested readily. When eating solid food rapidly, he sometimes gives the external esophagus a quick push with his wrist. His development has been normal in every way.

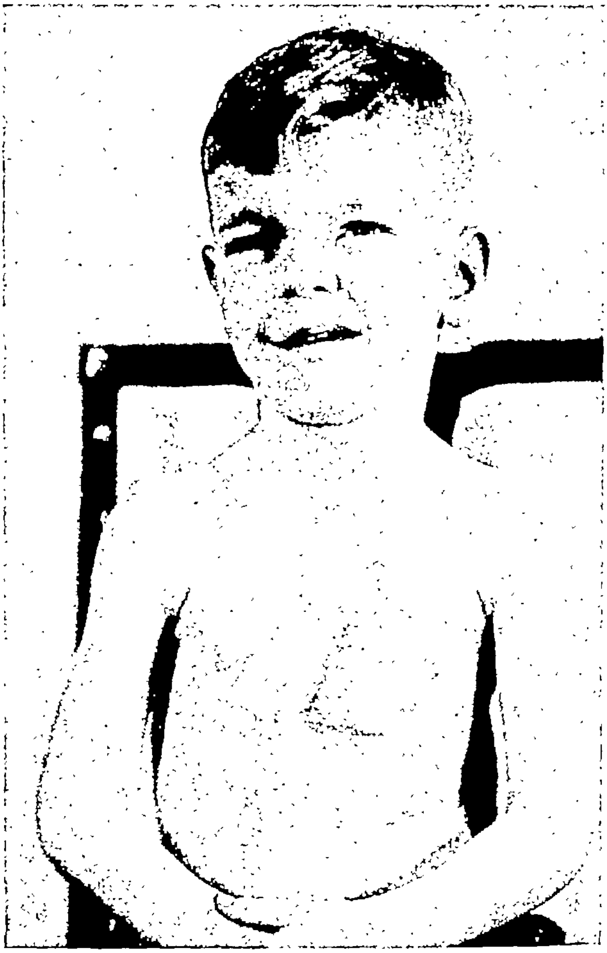

Fig. 5 (Case 1).—Patient at the age of 5 years, showing completed antethoracic esophagus.

CASE 2.—E. L., a boy (Case 7 in a previous communication[5]), was born on Dec. 8, 1945. On December 11, the diagnosis of atresia and tracheoesophageal fistula was made, and I immediately explored the mediastinum. The fistula was closed, and an attempt was made to form an end-to-end anastomosis, but the gap between the segments was too great. The lower segment was allowed to retract toward the abdomen, and the opening which had been made in the upper blind pouch was closed temporarily. (Stage 1, Fig. 1.) Three days later, the upper segment was exteriorized in the clavicular region (Stage 2, Fig. 1). Two days later, a transverse incision was made beneath the left costal margin and after section of the short gastric vessels, the left gastric artery and the vagus nerves, the lower segment of esophagus was drawn out of the hiatus and brought through a subcutaneous tunnel to a point to the left of the midline of the chest. (Stage 3, Fig. 1.) It will be noted that these three operative procedures over a period of a week accomplished the same anatomic result that five operations over a period of one year did in the first case (Fig. 6).

On March 18, a hernia of the left subcostal incision was repaired, and at the same time, a small pedicle was raised on the left side of the abdomen. This was only about 4 cm. in length, but it became necrotic in the middle. On August 14, an attempt was made to construct the dermic tube. As in Case 1, the skin of the adjacent chest wall was so loose that it could be brought over the tube without obvious tension, and no pedicle grafts were utilized. However, as a safety factor, relaxing incisions were made at each end of the tube. Again, the tube opened up, this time at both ends, leaving a tunnel between. This tube was dilated by passing olive-tipped dilators in both directions. On Jan. 6, 1946, a tubed pedicle from the left axillary region was used to cover the upper defect. Before healing was complete, a stricture developed in the end of the upper esophageal segment, and a small plastic procedure was carried out. The closure of the lower opening was delayed to permit occasional dilatations of the upper part of the tube, and on May 16, 1947, the lower defect was covered with a pedicle from the right side of the abdomen. The final operative procedure was on June 9. There had been a total of eleven trips to the operating room. Twice since then a small area of infection has developed in the suture at the medial and lower part of the tube. This has been followed by some leakage of fluid, but each time the opening closed spontaneously in two weeks. The tube has been water-tight for four months, and the child is eating normally (Fig. 7).

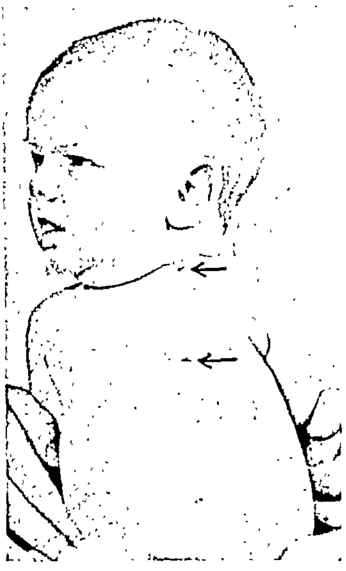

Fig. 6 (Case 2).—Patient at the age of 2 months. Upper arrow points to cervical esophagostomy which permits sham swallowing. The lower arrow points to the lower esophagostomy through which a catheter is inserted at feeding time. Note the absence of skin irritation from digestive juices, usually present with a gastrostomy.

There are other methods of constructing an antethoracic esophagus, many of which are reviewed by Yudin.[8] Ladd[9] constructed a long dermic tube to connect the cervical esophagostomy with a gastrostomy in the epigastric region in several patients. This was successful in at least one case, a girl who is now nearing her ninth birthday. Later, Ladd and Swenson[10] indicated that they had become discouraged with a method which involved the anastomosis of a skin tube directly to the stomach and recommended the use of a jejunal transplant to connect with a short dermic tube. With this plan, food completely bypasses the stomach, at least a theoretic objection. The group at the Children's Hospital in Boston have not remained enthusiastic about jejunal grafts, because in a recent article, Gross[11] had the following to say with regard to Yudin's method in adults: "In our clinic, attempts at similar jejunal transplantation in children (for the treatment of esophageal atresia) have been troublesome or have failed because of the shortness of the loop which can be brought up onto the chest or because of insufficient blood supply (and sloughing) of the limb."

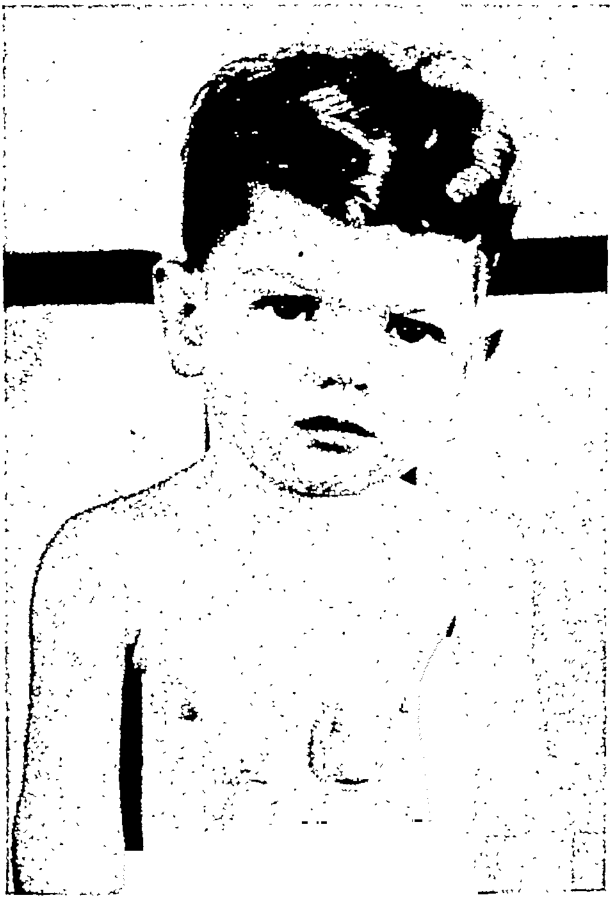

Fig. 7 (Case 2).—Patient at the age of 2½ years, with completed antethoracic esophagus.

Longmire and Ravitch[12] have devised a method of mobilizing a considerable length of jejunum by transplanting it into a skin tube raised in the left abdominal region, and later stepping this up into position on the chest. They were successful in one adult with a chemical stricture. The same method was used to connect the two esophagostomies of an infant with atresia who had had the preliminary stages thirteen months previously. This patient died at the age of 34 months, at which time there was still a fistula at the site of the esophagojejunal junction.

Rienhoff[13] recently reported two cases of atresia in which as a primary operation he had divided the esophagus at the diaphragm and mobilized the stomach so well that the cardiac end could be brought up through a skin tunnel and anastomosed directly to the upper esophagostomy without the intervention of a dermic tube. A patient operated on in 1941 lived one year and died of "staph. infection and diarrhea." His second patient also lived one year and died of diarrhea and a respiratory infection.

Still more recently, Sweet[14] described a new method by which he had completed the multiple-stage operation in a 21-month-old boy by bringing the mobilized stomach up through the chest and out to the left side of the neck through an opening made by resecting a portion of the left clavicle and first rib. He felt that the result was generally satisfactory, but the early reports indicated an unsatisfactory weight gain.

SUMMARY

In the management of two infants with atresia of the esophagus in whom the superior operation of direct anastomosis was not carried out, it was found to be advantageous to exteriorize the lower segment for feeding during the first year. Plastic procedures to connect this esophagostomy with the upper esophagostomy with a dermic tube were completed in one child when he was 27 months old and in the other when he was 18 months old. Disappointments encountered during the construction of the tubes probably could have been avoided by the use of pedicle grafts to cover the denuded areas. The function of the antethoracic esophagus in these cases appears to be as satisfactory as that obtained when more complicated procedures, such as jejunal transplantation, are utilized.

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DISCUSSION

DR. RICHARD H. SWEET, Boston.—If you will bear with me once again I will promise not to discuss another paper. I am much interested in Dr. Lam's experiences, which are about the same as we have had. The construction of an external esophagus from a skin tube is a tedious procedure. As he did, I have likewise tried pulling the stomach up for use as the lower end of the external esophagus. I would like to make a suggestion as to how this may be handled differently.

(Slides) This illustrates how I have handled these cases. A thoracic incision is made and the diaphragm is opened. The gastrostomy is closed, and the stomach pulled into the chest. After closing the thoracic incision, the proximal end or fundus of the stomach was brought up into the neck through a cervical incision after resection of a portion of the clavicle and a portion of the first rib as shown here.

This has been done in two infants, one about a year ago and one five or six months ago. They are getting along normally and beginning to develop. This is a one-stage operation and obviates the necessity for mechanical assistance. These children eat normally and are not aware of the fact that they have a stomach that has been pulled up into the neck.

DR. LLOYD H. MOUSEL, Washington, D. C.—If I may be allowed to digress for a moment, I would like to express my pleasure in learning at this meeting that the majority of major thoracic surgical clinics have abandoned the use of cyclopropane. During the past several years during which a number of deaths on the operating table have been reported, this agent was being used. Most of these deaths have been attributed to vasovagal or vagovagal reflex activity. It is my personal belief that deaths on the operating table due to cardiac standstill can be greatly reduced, if not entirely eliminated, if cyclopropane is not used.

In managing patients who are undergoing resection of the esophagus, positive pressure is maintained in the closed anesthesia system at all times, in order to prevent bilateral pneumothorax from developing should the opposite pleura be opened. Large openings into the opposite pleura are usually recognized by the surgeon and the lung can be kept inflated until the pleura has been closed. However, if a small opening is made into the opposite pleura, it may go unrecognized if positive pressure is not being maintained, and a complete pneumothorax may develop. Our patients are watched very closely on the operating table and great care is taken to prevent such a development.

Following each major thoracic surgical procedure, our patients are x-rayed on the operating table. If there is evidence of air in the pleural cavity, it is removed before the patient is returned to his room.

In our particular area, two cases have been reported in which the patient died on the operating table, due to accidental entrance into the opposite pleura. These patients were both undergoing lumbodorsal sympathectomy. In each instance the patient developed an obstructive type of breathing, and it was felt by the surgeon and the anesthetist alike that some obstruction to the airway had developed. Attempts were made to aspirate material from the tracheobronchial tree without results. Both patients deteriorated very rapidly and died before they were removed from the operating table. In each instance at post-mortem, it was found that the patient had died of bilateral pneumothorax. These are accidents that do not occur often and can be prevented by the use of constant positive pressure during the time the mediastinum is open.

DECORTICATION IN PULMONARY TUBERCULOSIS INCLUDING STUDIES OF RESPIRATORY PHYSIOLOGY

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S. WELLES, M.D., SARANAC LAKE, N. Y.

THE historical identification of the decortication operation has been previously made by the late Dr. Fraser Gurd[1] and will, therefore, not be further considered. The present revival of interest must be dated from the reports given at the Detroit Meeting of this Association in May, 1946, on chronic traumatic hemothorax and empyema.[2-4] The purpose of this presentation is to relate experiences gained thus far in exploring the applicability of decortication to the inexpansible tuberculous lung. The eight cases to be reported upon is a small number for a series, but serve as a basis for suggesting a method of study from which helpful conclusions might be drawn. The operative technique has been established and need not be reported here.

The problem of hemothorax and pyothorax with or without air present is not entirely comparable with that of the inexpansible tuberculous lung and empyema. Common to both is the objective of obliterating the pleural space by re-expansion of the lung. The fundamental pathology and method of treating the underlying pulmonary disease are not the same. The treatment of the pleural complication in the former generally represents an end point. In the latter, the pleural problem is always secondary to the parenchymal disease which is the chief consideration.

For the prime purpose of obliterating the pleural space, thoracoplasty alone has proved satisfactory in many cases. Upon occasion, in the presence of bronchopleural or pleurocutaneous fistulas, the addition of a Schede or myoplastic operation has been required. While thoracoplasty accomplishes a twofold purpose, permanent collapse of the diseased lung and obliteration of the pleural space, it does so at the sacrifice of good functioning lung in certain cases. For this reason and because of the desire of avoiding more mutilating surgery, the decortication operation suggests itself. The comparison of results must be made between the two procedures. In attempting to bring about re-expansion of a lung in which tuberculosis has been successfully treated, one must be mindful of possible harmful effects. The question of risk is naturally posed. This must be weighed against the hazard of long-sustained pneumothorax. Once it has served its purpose, it is desirable to abandon the pneumothorax, as expected reversibility is one reason for its application.

That decortication is possible in cases of pulmonary tuberculosis has been demonstrated in several reports: Gurd,[1] Mulvihill,[5] Curreri and Gale,[6] and Lam.[7] It was recognized at the outset that because of variable extents of parenchymal disease for which the lungs were collapsed, re-expansion and improved function

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might not necessarily follow. In anticipation of such problems a cooperative study was outlined with Dr. George Wright, of the Trudeau Foundation Physiology Laboratory.

The starting point for considering treatment of a disease or complication is a knowledge of its pathology. The detailed pathology of tuberculosis need not be considered here. The effect of long-sustained collapse on the underlying lung is of importance and serves to answer the question, "Can the lung be re-expanded?" The morphology of collapsed uninvolved pulmonary tissue as given by Vorwald[8] is as follows: "There is collapse of the alveolar spaces, the walls of which are approximated, but the architecture of the lung is not obliterated; there is histocytic proliferation of the alveolar walls and in the perivascular and peribronchial spaces with a slight increase in connective tissue; the blood vessels are more or less tortuous and the capillary spaces dilated; the lymphatics are little altered but may be dilated; the degree of connective tissue proliferation is not irreversible." The experimental work of Bruns,[9] and the studies reported by Gardner[10] and Pinner[11] are the basis for the conclusions that "fibrosis in a collapsed lung is the result of inflammatory processes and not of the collapse per se." It is upon this basis that re-expansion of the uninvolved portions of the lung is expected. The anticipated degree of re-expansion, judged by the prepneumothorax extent of disease does not necessarily follow. Gardner stated that "extension of disease within the compressed lung may proceed by way of the air passages and, to a more limited degree, through the lymphatic channels. Hematogenous metastasis is probably rare." These data serve as a working base line for approaching the present problem.

The cases selected for treatment were those in which there was evidence of failure of obliteration of the pleural space by the lung. All measures to have the lung re-expand and obliterate the pleural space had failed. There was no question that perhaps in time this might yet occur. Cases with advanced stenosis of a major bronchus were excluded. In these it was felt that re-expansion was probably impossible and undesirable for fear of increasing the broncho-parenchymal bed distal to stenosis and risking suppuration. In five cases (Cases 1, 2, 3, 4, and 5) the duration of the pneumothorax was over years and the parenchymal disease was considered arrested, the sputa having been negative. Seven of the eight patients had fluid present which was positive for tubercle bacilli, two showing frank pus. Two patients (Cases 6 and 7) had active pulmonary disease, with cavities and positive sputa. In these thoracoplasty was done first to collapse the disease-containing parenchyma before decortication was undertaken. In Case 8, there was a history of pleurisy with effusion twenty-seven years before with recurring symptoms in 1942 and 1947. There was no clear-cut history of pulmonary tuberculosis until 1947, when a small antrum was noted on the contralateral side (left), the sputum and fluid were both positive, and intermittently a bronchial fistula could be demonstrated.

The history and all available films of each case were carefully reviewed. A careful fluoroscopic survey as well as a study of inspiration-expiration films indicated the present mobility of the lung. The factors of compensation, intrapleural pressures, frequency of refills and the speed of re-formation of fluid were noted. All were bronchoscoped before operation and special respiratory studies were also made, both before and after decortication. In two cases preliminary bronchograms as well as two other postdecortication bronchograms were made.

From these preliminary studies a decision was made as to whether a thoracoplasty would be done before undertaking the decortication. In two of the arrested cases (Cases 2 and 3), it was elected to do two stages of thoracoplasty because of the extensiveness of the tuberculosis before pneumothorax. It was reasoned that insufficient good lung remained to anticipate enough re-expansion to fill the large pleural space. In Case 2 there was some arterial oxygen desaturation observed after exercise on a motor-driven treadmill. Decortication resulted in decreasing the pleural space and some increase in lung volume but no functional improvement. It was, therefore, felt that additional thoracoplasty should be done to aid in obliteration. This has recently been done and there is evidence that a satisfactory result will ensue. In Case 3 decortication obliterated the small residual space. It is expected that no functional change has resulted. In Case 1 the patient had been treated with an emergency thoracotomy drainage because of fulminating uncontrollable mixed-infection empyema and bronchopleural fistula. Subsequent multiple-staged thoracoplasties were done to obliterate the large empyema space further to control the toxemia. A residual pleural space and cutaneous fistula at the site of the thoracotomy wound remained. Decortication was carried out in preference to a Schede operation; the lower lobe was mobilized and a good result obtained. The patient was discharged as well and has been working for a year. In Cases 4, 5, and 8 primary decortication was performed. In Case 4 ultimate complete obliteration of the pleural space was accompanied by further desaturation of the arterial blood; the detailed report of this case will be given by Dr. Wright.[12] Incomplete obliteration of the pleural space with fluid forming in the presence of high negative intrapleural pressures resulted in Case 5, with no significant change in pulmonary function. The primary pleurisy with effusion case of twenty-seven years' duration (Case 8) has almost complete obliteration, but it is too recent postoperatively to evaluate.

In the two cases in which thoracoplasty was done for active pulmonary disease, decortication resulted in obliteration of the remaining pleural space, thus obviating the need for further thoracoplasty. These two are of additional interest regarding findings at the time of decortication. In Case 6, a completely collapsed lower lobe was found covered with peel. A second review of the films indicated that this might have been anticipated. With removal of some peel, tubercles were seen and it was elected not to complete the operation except over the uninvolved portion of the upper lobe. After decortication the lower lobe of Patient P. R. in Case 7 appeared slate-blue in color and, though the lobe was distended with positive pressure, the color did not change to the usual pink. The significance of this finding is not clear.

In all except the case with the twenty-seven-year history, the removal of the visceral pleural peel was relatively easy to accomplish with minimum trauma to the lung. In this case much sharp dissection was required and a strawberry-like appearance was seen on the pleural side of the cortical layer. In all cases there were areas where the peel was most adherent which were considered healed foci. After a brief trial at removal these were circumvented and allowed to remain in patches. Studies before operation showed in all cases some reduction in respiratory function proportional to the extent of the pulmonary problem. Those studied postoperatively thus far have not shown any significant gain in function as contrasted with one nontuberculous traumatic hemothorax case to be discussed elsewhere.[12]

There were no spreads of disease, but it must be stated that streptomycin was used for short periods of time, both pre- and postoperatively. The significance of this therapeutic adjunct is not known. All cases were on continuous suction for from forty-eight hours to five days. There were no recurring empyemas and all wounds healed by primary union.

In addition to the above-noted results there are some points which require further consideration. What of the re-expanded lung? Will it be normal healthy lung, or one showing compensatory dilatation or, one step further, true pathologic emphysema? The lung may be ventilated with poor circulation, or have good circulation and be poorly ventilated. There may be satisfactory readjustment or the development of a shunt between unaerated and aerated blood. The volume of lung for re-expansion is unknown because the amount of interstitial fibrosis cannot be determined. The amount of disease seen in roentgenograms is by no means an index of all that is present. With healing and scar formation of the diseased areas there are local compensatory changes in the surrounding lung tissue. In addition, there is the probability that, under partial pneumothorax at least, compensatory changes in the lung have already taken place. Hence, some limit of distensibility may have been reached and may account for the failure of further expansion. It is known that in certain cases where no previous pleural complications are suspected transudate fluid forms to take up the space the lung cannot occupy. In these, perhaps the limiting membrane may be a secondary formation rather than the cause of limited expansibility. In several cases bronchography has been done and there is some suggestion that it may be helpful in estimating the above-mentioned changes. How long after decortication does it take the lung to return to normal? For the present the answer cannot be given. These are the observed facts: (1) Immediately upon removal of the peel, the lung becomes distensible by positive pressure on the anesthetic bag with an intratracheal tube present; (2) increase in lung volume is noted within several days as seen on postoperative roentgenograms; (3) the diaphragm is seen to move in a normal direction. Recheck bronchospirometry is planned and in one nontuberculous case progressive improvement in function was recorded. That some time must elapse before the uninvolved portions of the lung become restored might be expected in considering the previously described histopathology. A certain lapse of time was required to produce these changes and should be necessary before function is restored. This is certainly true of other immobilized parts of the body. Regarding the peel that is left behind, since the etiology of the fluid is generally tuberculous and since these effusions and empyemas are clinically known to regress and recur, the question arises as to possible recurrence in the decorticated case. Insufficient time has elapsed in this group of cases to permit making a statement. It might be of some significance, however, that histologic examination of these peels show nonspecific scar in the older cases and tubercle formation in the more recent ones. Further, the fluid was examined and, in cases previously positive on smear and culture, has been negative by culture postoperatively. The parietal peel was more difficult and was only partially removed in some cases. Experience indicated that a plane of cleavage was easier to develop at the endothoracic fascia, where bleeding was most profuse, than between pleura and cortical layer. Whether or not it is essential to remove the parietal peel is not known. There is some suggestion that this may not be entirely necessary. For example, it is well known that, in certain chronic empyema cases, apparent resolution takes place so that no evidence of the previously designated "thickened pleura" is roentgenographically demonstrable (Case 9). In addition, the previously observed compensatory factors of flattening and immobility of the chest wall are replaced by normal contour and movement in the presence of a good functioning

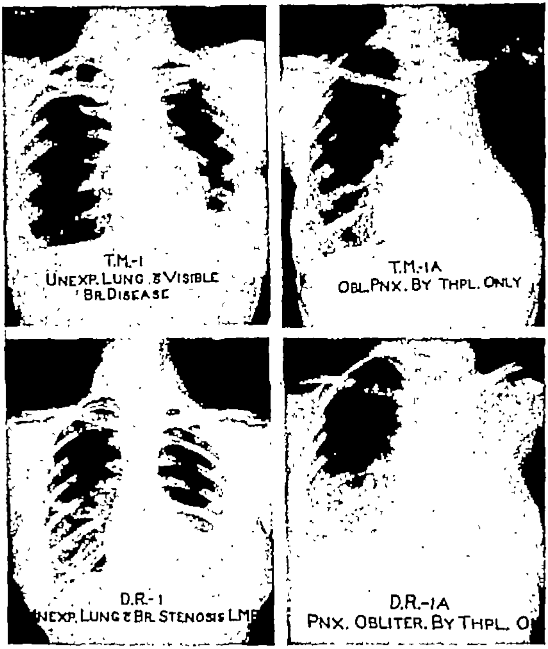

Fig. 1.—T. M. *1*, showing unexpandable left lung, pneumothorax space with fluid present. No evidence of bronchial disease by bronchoscopy. T. M. *1A*, showing obliteration of entire pneumothorax space by thoracoplasty only. D. R. *1*, showing almost total collapse of left lung, large pneumothorax space, bronchial stenosis of left main bronchus seen by bronchoscopy. D. R. *1A*, showing satisfactory obliteration of left pleural space by thoracoplasty alone.

lung. Further, some of the decorticated-hemothorax cases did not have the parietal peel removed and are reported as restored to normal function. Of particular consideration in these tuberculous cases is the fact that the volume of lung for re-expansion is an unknown quantity. A completely intact hemithorax may not be required because of limited distensibility of the lung and the pleura may be again thickened as a compensatory factor following re-formation of fluid.

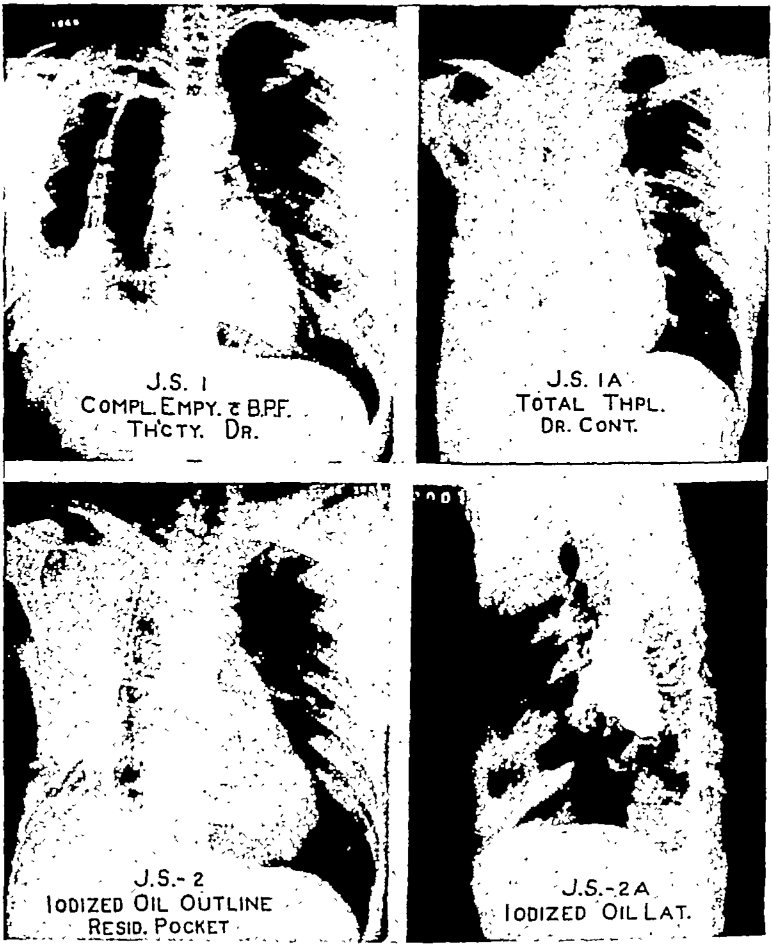

Fig. 2 (Case 1).—J. S. *1*, film showing partially collapsed lung with markedly thickened peel on both pleurae, thoracotomy tube drainage because of bronchopleural fistula. J. S. *1A*, film showing total thoracoplasty collapse, tube drainage still present. J. S. *2*, film showing outline of residual empyema pocket in the posteroanterior projection. J. S. *2A*, same as above in the lateral projection.

A further point for longer term observation is the possibility of development of a reactivation of disease at a later date in the re-expanded lung. Is there a possibility that this distended or emphysematous lung is made more vulnerable to the development of active disease? There is some suggestion, but not proof, in some of the obliterating so-called "re-expanded pneumothorax" cases. With mediastinal shift to the obliterating side and increased size of the contralateral lung new disease or reactivation of stable foci have been noted to develop. This point alone is of little moment, but in reinducing collapse of the nonex-

SERUM PROTEIN LEVELS AND CALCULATED OSMOTIC PRESSURES IN TUBERCULOSIS: I. CORRELATION OF THESE VALUES WITH THE SEVERITY OF THE DISEASE*

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THE present investigation of serum proteins in pulmonary tuberculosis was instigated more or less directly by experimental observations on dogs[1] in which acute hemorrhage was found to cause pulmonary edema. A coincidental finding of these experiments on hemorrhage showed that acute reduction of the total serum protein by as little as 0.8 Gm. to 1.0 Gm. per 100 ml. of serum as a result of the intravenous administration of physiologic sodium chloride would also produce edema of the lung. The pulmonary edema was demonstrated in both of these animal experiments by dehydration procedures and by histologic section of the lungs, and it was a consistent observation even when the moisture increase was too little to be clinically apparent.

It is recognized that the serum proteins are of great value in maintaining the normal osmotic pressure of blood, and that the osmotic pressure, in turn, is of importance in regulating the equilibrium of tissue fluids. Because of the possibility that edema of the lung may contribute to the pathogenesis of pulmonary disease, it seemed possible that a lowered serum protein level might be a detrimental factor in any infectious disease of the lung by virtue of the pulmonary edema it could induce. The concept received encouragement by the favorable clinical results observed with plasma therapy in several children with pneumonia resistant to other treatment.[2] In these patients it was found that the total serum protein, the serum albumin, the albumin-globulin ratio, and the calculated osmotic pressure were all low; and intravenous plasma was given these patients for the purpose of restoring the low serum protein values to the normal, hence raising the osmotic pressure and reducing lung edema.

It seemed possible that in pulmonary tuberculosis the presence of edema of the lung could also be a contributing factor to the pathogenesis of this disease, and that edema might be present more frequently than is realized in association with lowered serum proteins. Seibert and Nelson[3] demonstrated by electrophoretic patterns with sera from animals with experimental tuberculosis that there was a consistent decrease in the serum albumin level and a rise in beta-globulin, which could be detected as early as ten days to two weeks following the infective inoculation. In several cases of human tuberculosis these authors noted abnormally low serum protein values which seemed to parallel the clinical

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*Cooperative study, Sunshine Sanatorium, Kent County, Michigan; and Michigan Department of Health Laboratories, Western Michigan Section, Grand Rapids, Mich.

course of the disease. It could well be, therefore, that depletion of the serum proteins with the resultant lowering of the osmotic pressure of the circulating blood could produce sufficient pulmonary edema to be a factor in the course of pulmonary tuberculosis and a stumbling block to the cure of this disease. It could also be postulated from known facts concerning the role of the serum proteins in maintaining normal fluid equilibrium between the vascular system and the pulmonary alveoli, that elevation of the serum protein levels could attract fluids back to the vascular system and thereby dehydrate an edematous lung and make tuberculous spread more difficult.

Therefore, the whole question of the relationship of serum protein levels to the progress of pulmonary tuberculosis seemed well worth investigating, and a study was started early in 1948 for the purpose of answering the following questions:

1. Can we demonstrate consistently an alteration of the serum protein levels and of the calculated osmotic pressures in patients with pulmonary tuberculosis?
2. If there is an alteration, is it in proportion to the severity of disease?
3. Can the serum protein levels in a patient be influenced by intravenous plasma, or by the oral administration of protein hydrolysates?
4. Does high protein therapy influence the clinical course of pulmonary tuberculosis?

The present report has to do with the first two questions.

METHODS

Clinical classification and selection of patients.—

The patients were classified by roentgenogram findings according to the standards of the National Tuberculosis Association into minimal, moderately advanced, and far advanced tuberculosis; and according to their clinical status, into A, B, C, and D groupings, group A indicating patients with no apparent symptoms of illness, and group D representing those critically ill. The clinical status was based on such items as temperature, pulse, weight loss, appetite, expectoration, and positive or negative sputum findings. The classification of each patient was reviewed at least every six months, and for the purpose of this study those patients were excluded who had complicating diseases such as severe diabetes, nephritis, or nephrosis. The classification of the 152 patients comprising the study group is given in Table I.

TABLE I. SANATORIUM PATIENTS STUDIED SHOWING THE NUMBER IN EACH CLINICAL CLASSIFICATION*

CLINICAL STATUS	STAGE OF TUBERCULOSIS			
	MINIMAL	MODERATELY ADVANCED	FAR ADVANCED	TOTAL
A	0	3	0	3
B	11	51	15	77
C	0	18	31	49
D	0	4	19	23
Total	11	76	65	152

*According to National Tuberculosis Association definitions.

sure which regulates fluid equilibrium between the vascular system and the tissues. In this connection the discussion by one of the authors[7] in relation to lowered serum proteins associated with spinal cord edema in poliomyelitis is relevant. Of the serum proteins, the albumins are the smallest molecules and are the most copious. In normal plasma, the albumins make up over 60 per cent of the protein constituent and, according to several physiologists, including Best and Taylor,[8] they are responsible for nearly 80 per cent of the colloidal osmotic pressure exerted by the plasma. Therefore, any appreciable change in the albumin content of the plasma will have an immediate effect upon the osmotic pressure which this plasma is capable of exerting. Loss of positive osmotic pressure as a result of a reduction of the albumin content, in which there is usually a reversal of the albumin-globulin ratio, will induce a transudation of the small albumin molecules, as an albuminous fluid, to the tissues and into the alveoli. Such decrease in serum albumin caused either by accelerated protein catabolism or disturbed protein synthesis and occurring as one of the systemic manifestations of disease, would lower the plasma osmotic pressure and accentuate the edema of the lung. Conversely, if the serum albumin and osmotic pressure were raised, the process might be reversed; the lung might become less moist and less likely to encourage the spread of the disease tuberculosis. The findings would seem to justify further experiments directed towards raising the albumin level and hence the osmotic pressure, by intravenous plasma or the oral administration of protein, and a study of the effect of such an alteration on the clinical course of the disease.

SUMMARY AND CONCLUSIONS

The results of serum protein studies in 152 patients with pulmonary tuberculosis have been reported.

The most significant alteration appeared to be in the serum albumin fraction, and, in general, it was found that the more severe the disease, the lower was the albumin level.

It is believed that serum protein levels deserve greater attention in the evaluation of pulmonary tuberculosis. Further studies are in progress to investigate the value of intravenous plasma and oral protein hydrolysates in the therapy of selected tuberculous patients.

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BRONCHIAL ADENOMA*

NOMENCLATURE AND PRESENT METHODS OF TREATMENT

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BRONCHIAL adenoma has been recognized for many years. In 1937, for the first time, Zamora and Schuster[1] reported an adenoma which presented evidence of malignancy by local invasion. By 1943 several cases of distant metastasis,[2, 3, 4] picked up microscopically and incidentally, were reported. In 1943, Anderson[5] reported a macroscopic metastasis to the liver. Gordon[27] reported removal of the left lobe of the liver for tumor identical with a bronchial adenoma removed earlier in the same patient. Similar reports[6] followed. Adams[25] reports death due to cerebral metastases in a woman known also to have liver metastases from bronchial adenoma. From the time of the initial report in 1937 to the present time there has been discussion as to whether these tumors are benign or malignant. Since the lesion is relatively uncommon, facts which have become known about it have been disseminated only slowly. Generally, treatment has not varied a great deal because those not treated initially by surgery usually come to surgery later as a result of complications. A point we would like to make is that surgical resection, or at least exploration, is generally indicated as primary therapy for these tumors. It is commonly accepted that they are not of a degree of malignancy to be compared with that of bronchiogenic carcinoma, but some still deny that they are malignant at all.[7] As the subject continues to be one for study, efforts are being made to correlate clinical course with pathologic findings, and to try to establish some concrete basis on which to determine what treatment of these should be.

One opinion as to the origin of these tumors is that they arise from glands of the bronchial mucosa. Stout[9] and Haemperl[10] demonstrate certain cells in these glands by special staining which they call "Onkocytes." These they believe to be the source of bronchial adenoma. Womack and Graham,[3] and Harris and Schlattenberg[11] feel that the tumors are due to persisting embryonal bronchial buds. A resemblance to fetal lung tissue is noted by Churchill,[13] who suggests that they are vestigial pulmonary lobes growing within the bronchus. Similarity of some of the tumor cells to lymphocytes has been described by certain authors[12] who proposed that to such tumors the term lymphoid or angiolymphoid be applied.

Only one hypothesis in the discussion of possible sources of these tumors seems to be accepted with something approaching unanimity of opinion: these tumors, though considered to be epithelial, are separated character-

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From the National Naval Medical Center, Bethesda, Maryland.

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*Statements made in this paper are those of the author and do not necessarily represent policies of the Veterans Administration or U. S. Naval Bureau of Medicine and Surgery.

istically from the surface epithelium of the bronchus, and it would appear that they do not take their origin from the surface epithelium, but probably from bronchial glands.[5, 14-18]

Clinically, the symptoms resulting from bronchial adenoma can be ascribed in the majority of cases to complication more frequently than to the tumor directly. Cough, hemoptysis, and expectoration are common, but probably arise because of a condition brought about by the obstruction of the bronchus—atelectasis, bronchiectatic changes, and so on. Pneumonia, pleurisy, or even empyema may be the presenting clinical picture, or the patient may complain of mild chest pain, dyspnea, fever, weight loss, or wheezing. Severity of symptoms will depend for the most part on severity of the secondary conditions, and these will be influenced as much by the location of the tumor as by its size. Physical findings will be conspicuous by their absence unless secondary developments have appeared, in which case the findings will be those of the secondary condition. This is worthy of emphasis because cases may go undiagnosed because of a paucity of findings or because bronchial adenoma is not suspected. Treatment without proper diagnosis may thus be unnecessarily prolonged. In reported cases treatment for bronchiectasis, pulmonitis, and so on, has extended over long periods before bronchial adenoma has been suspected as the underlying cause.[19] Certainly, in some of these cases early removal of the source of the obstruction might be expected to relieve the condition before irreparable damage has occurred.

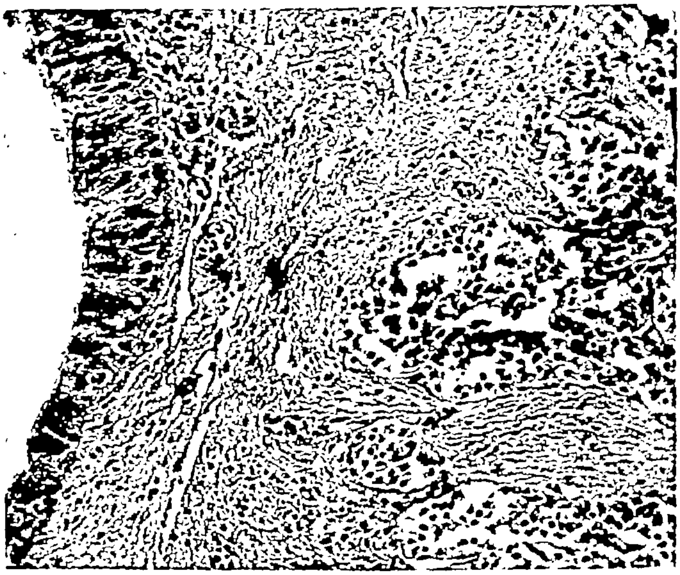

Fig. 1.—Carcinoid adenoma (×200), showing relationship to bronchial epithelium.

Abnormalities demonstrable by roentgenogram[20] include: demonstrable tumor, atelectasis, inflammatory infiltration, or any combination of these. In one report the only finding was unilateral emphysema in expiration, due to a ball-valve type of obstruction. The routine roentgenogram of the chest was normal. In the series of cases reported by Holly,[18] a tumor was visible in 20 per cent, atelectasis in 40 per cent, and inflammatory infiltration in 23 per cent. Tumor was not demonstrable by roentgenogram in any of the cases included in this report.

Bronchoscopically, the tumor appears as a smooth, red, rounded tumor, usually with a broad base. Bleeding appears with minimal trauma, and at times may be a source of considerable discouragement when biopsies may be desired. Tendency to bleeding of serious proportion is shown by reported cases in which death has resulted from massive blood loss and shock,[22, 23, 24, 42] or the patient drowned in his own blood when the respiratory passages were suddenly inundated. The majority of bronchial adenomas occur in the primary passages, and they seem to be more frequently located in the right main lower lobe bronchus. With a tumor located in this position prolonged bronchoscopic treatment and observation may be enticingly convenient.

The presence of such a lesion, of course, requires biopsy for diagnosis. The pathologist also has his problem with these tumors. Without, for the moment, going into the pathologic ramifications, it may be said that not infrequently it is very difficult to distinguish some of these from small, round-cell carcinomas of the bronchus, which are highly malignant. Many instances have been reported in which distinction has been made only on subsequent review of the section or after a patient's clinical course failed to corroborate the prognosis. On frozen section in many of these, such differentiation is virtually impossible. This should give further impetus to efforts directed toward clarification of characteristics of these tumors and toward attainment of a common ground of understanding regarding them, for a decision made during exploratory thoracotomy may be greatly influenced if this fact is borne in mind. An adenoma with mediastinal metastases would probably offer an excellent opportunity for cure, whereas a small, round-cell carcinoma with such metastases would probably not justify even an attempt.

The following case reports will illustrate several points we feel should be emphasized:

CASE REPORTS

CASE 1.—P. H. D., a 25-year-old white man, was admitted on Nov. 14, 1942, with a complaint of hemoptysis of about half a cup of blood daily for about a week. A month prior to this he had had bronchopneumonia. After discharge from the hospital he noticed a "tickling in the throat," and on coughing and clearing his throat he would bring up one or two teaspoonfuls of bright blood. The cough was otherwise nonproductive. There had been weight loss of twenty pounds in the period preceding the present illness, but this was felt to be related to factors other than his physical illness. Physical examination was not remarkable except for a tiny ulcer found on one of the vocal cords. Bleeding was considered not to be coming from this. Bronchoscopy disclosed a tumor in the left lower bronchus which bled on slightest instrumentation. Frozen section was interpreted as "Oat-cell Carcinoma of the Bronchus," but when permanent sections were examined the diagnosis was changed to "Benign Adenoma *of the Bronchus*." On Dec. 1, 1942, the tumor was removed locally through the bronchoscope. Healing was rapid and satisfactory. A month later slight fullness was visible at the site. Four months later this had progressed, and it was apparent that the tumor was still present. In August, 1943, it was again removed through the bronchoscope at Memorial Hospital in New York. In November, 1943, it was once more visible and was cauterized through the bronchoscope. There was no further trouble until early 1948 when further symptoms developed. In May, 1948, a pneumonectomy was performed at the Ochsner Clinic. Since that time the patient has been well except for minor dyspnea on exertion. In this case resection was undertaken after five years of prolonged bronchoscopic treatment and observation. It would appear that the final result could have been achieved five years earlier if surgical exploration had been carried out.

CASE 2.—H. K. M., a 37-year-old white man, was admitted in March, 1949, complaining of persistent, mild, relatively nonproductive cough. There were no other symptoms, and general health was good. There had been no weight loss. Chest roentgenogram showed atelectasis of the right middle lobe, but repeated studies failed to disclose the cause of the atelectasis. It was felt that bronchogenic carcinoma was the most likely possibility. The patient refused operation. On Sept. 6, 1949, he returned with the same picture, which had persisted unchanged. General condition remained good. On Sept. 10, 1949, the right middle lobe was removed. A frozen section was interpreted as small, round-cell carcinoma, and total pneumonectomy was done. Enlarged hilar nodes were found and removed. The patient made a good recovery, and was discharged to his home on Sept. 26, 1949. In December, three months later, he was preparing to return to his regular work. Paraffin section showed the tumor to be bronchial adenoma of the carcinoid type. Three nodes were removed, which were not contiguous to the bronchus; these contained tumor which duplicated the pattern of that found in the parent growth. Here metastasis had occurred even with relatively short clinical duration of illness. At surgical exploration it was possible to remove nodes already containing tumor which might never have been resectable after bronchoscopic removal of the parent growth followed by prolonged observation.

CASE 3.—W. K. K., a 27-year-old white man, was admitted July 30, 1947, with a history of fever, nonproductive cough, anorexia, and vomiting for five days. He developed pain in the right chest which was aggravated by breathing, and on the day prior to admission had dyspnea and slight cyanosis. On admission a diagnosis of lobar pneumonia of the right lower lobe was made, with overlying pleurisy. This was confirmed by roentgenogram, but remark was made that there was a large atelectatic factor. On Aug. 6, 1947, bronchoscopy disclosed a mass which bled very easily, obstructing the right lower lobe bronchus. Frozen section of the biopsy was diagnosed as small, round-cell carcinoma. Later, permanent sections were interpreted as "Malignant Adenoma." On Aug. 14, 1947, a right pneumonectomy was performed when it became evident that the tumor not only filled the lower lobe bronchus but was also encroaching upon that of the right middle lobe. Several hilar nodes were removed but showed no tumor. The patient was discharged Sept. 15, 1947, in good general condition. The final pathologic report was "Bronchial Adenoma, with involvement of two contiguous peribronchial nodes." Bronchiectasis and pneumonitis were also present. In November, 1949, the patient reported that he had morning cough with which he brought up a considerable amount of "phlegm." He had had no significant cough thereafter during the day. This might indicate some mild bronchiectatic process in the opposite lung. It points up the danger of delay in treatment which may permit infection to become established in the opposite lung. The case demonstrates again that removal without surgical exploration is not adequate. Routine chest roentgenogram was clear in August, 1949.

CASE 4.—C. W. H., a man in his late forties, was admitted in May, 1944, for repair of a small right inguinal hernia. On questioning, he remarked that it had developed because of a bad cough he had had for about a year. Chest studies were carried out, and he was found to have complete obstruction of the left lower lobe bronchus with atelectasis of the lobe. Biopsy was interpreted as "Basal-cell Carcinoma, probably arising in a benign adenoma." A left pneumonectomy was carried out, the patient made a very satisfactory recovery. The operative specimen showed a tumor 4 cm. in diameter in the main bronchus of the left lower lobe. No metastases were evident, but there was an extensive infiltration through the bronchial wall into surrounding lung tissues. The patient, at the time of this writing, is progressing very satisfactorily. He weighs approximately fifteen pounds more than he did prior to the operation. There is a mild residual cough, believed to be associated with accumulation of secretion in the blind bronchial stump. Dyspnea is present only on exertion and is surprisingly little. On review of the sections this tumor was found to fall into those which we call the mixed tumor group, also called, among other things, adenocystic basal-cell carcinoma of the bronchus, or cylindroma. This type is considered by many to be more malignant, metastasizing more frequently than the carcinoid type.

Until 1937 little question was ever raised regarding the supposedly benign nature of these lesions, and major surgical procedures were undertaken only when necessitated by complications. Since then the various improvements in surgical techniques and methods of treatment and the continual reports indicating malignant characteristics in these lesions have aroused in many the feeling that surgical exploration, usually with resection, should be the preferred method of treatment. Secondary factors or unassociated conditions may, of course, interfere with the carrying out of the most desirable therapy.

The relatively few tumors not accessible to bronchoscopic instruments must be treated by other methods. Since the majority are in the primary passages and are commonly in the lower lobe bronchi, however, there is some choice. They may be subjected to biopsy, fulguration, desiccation and cauterization, or, rarely, excision through the bronchoscope. In 1940, Eloesser[29] reported the use of posterior bronchotomy as a means of approaching and removing these tumors without sacrificing the lung. This was again mentioned in 1947 by Goldman,[30] as being used in one case. Langston and Fox,[31] in 1948, described a technique of posterior bronchotomy for the treatment of this condition, but emphasized that such approach is limited in its use and is possible only in selected cases. Lobectomy may offer the best attack, and pneumonectomy will be necessary in some. Complicating infection may prevent treatment directed toward the tumor itself.[19] In some instances a combination of types of treatment may be called for. Recurrence after bronchoscopic removal should be sufficient indication for surgical exploration and resection, if possible. Inability to remove a tumor surgically would call for palliative measures by the bronchoscopic route. The bronchoscope may permit relief of obstruction, evacuation of suppurative material, and may at times allow preservation of some of the pulmonary tissue in subsequent removal of the tumor at surgical exploration.

Series have been reported to support the views of those favoring bronchoscopic treatment,[32] and others to support the views of those favoring exploration and resection. It must be admitted that statistical differences are not striking.[33] This is not remarkable, however, for the tumors are slow-growing, metastasize only occasionally, according to present reports, and five-year cures would be an unsatisfactory criterion. On the other hand, the staunchest of those claiming these tumors to be benign admit that they invade locally, invasion of the bronchial wall being considered characteristic.[34] Extension to the mediastinum is admitted generally, although some feel this is simple extension, while others consider it often frank metastasis. In 1945 Moersch, Tinney, and McDonald[35] stated: "We have never seen a case with metastasis, and this ultimately distinguishes between this ('adenoma'—'cylindroma' not included) and bronchogenic carcinoma." Van Hazel, Holinger, and Jensik[36] conclude that "adenomas" are extremely slow-growing and locally invasive, whereas the "cylindromas" "met all the criterion of malignant neoplasm."

The former group reported six cases of "cylindroma" with the opinion that these resemble mixed tumors and present a different appearance from "Adenoma of the Bronchus." Others, however, feel that all these tumors can only be considered as one group—adenoma. Holly,[18] in reporting thirty-nine cases, felt that it was possible to distinguish two groups, the "Carcinoid" and the "Mixed Tumor Type." Laff and Neuberger[37] recognized those with a "carcinoid-like" pattern and those with a "cylindromatous" pattern. However, both groups described by Laff and Neuberger are included in one group by others, and the "cylindroma" described by some falls into the mixed tumor group of Holly.

Such confusion tends to disorganize any concerted attempts at clarification and certainly would discourage many of those more likely to be concerned with the early recognition of the condition and generally the early treatment of the average case—the general practitioners.

There are objections to be raised to most of the terms already in use. Carcinoid has long been applied to tumors of the appendix and the gastrointestinal tract which show argentaffine granules. Tumors of the bronchus which resemble these morphologically have not shown such granules except in isolated and unconfirmed instances. Carcinoid-like was coined by one group to circumvent this, but seems redundant. "Adenoma" is a term which has long been applied to these tumors as a whole, and to the average medical group continues to mean just that. To take this term and apply it now to certain types as distinguished from others in the same category is to bring about more misunderstanding. The term cylindroma, which at present seems widely in favor, has brought possibly more confusion than any of the others.[5] One attempting to study these growths must, of necessity, familiarize himself with what is meant in each case when the term is used, but if further knowledge in the field is to be disseminated, consistency must be maintained. According to Dorland's Medical Dictionary, a cylindroma is a tumor in which the *stroma* has the form of elongated, twisted cords of hyaline material. According to the caption of a photograph accompanying a recent article on the subject[36] the cylindroma (bronchial) is one in which the "acini" are likened to "cylinders." It is the concept of the cylindroma as described by these authors which seems to be most commonly held. Again, in *Anderson's Pathology*[38] "The term cylindroma refers to the hyaline tubes and cylinders of the stroma." Similarity of the "cylindroma" to epithelioma adenoides cysticum has been noted. According to Menetrier, in 1927,[39] Billroth was the first to apply this term, and did so when a tumor which was squeezed produced rounded long masses which he thought resembled cylinders. In 1932, Stout[10] stated: "The term cylindroma is no longer used, and rightly so; because it describes simply a morphological variation and not an entity. The mucinous "cylindromas" should be called mixed tumors; the hyaline "cylindromas" (skin) should be called basal-cell

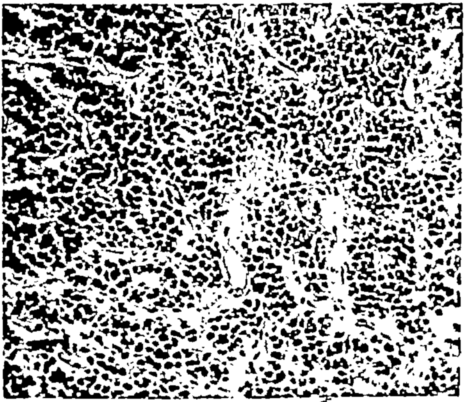

Fig. 2.—Carcinoid adenoma (×200).

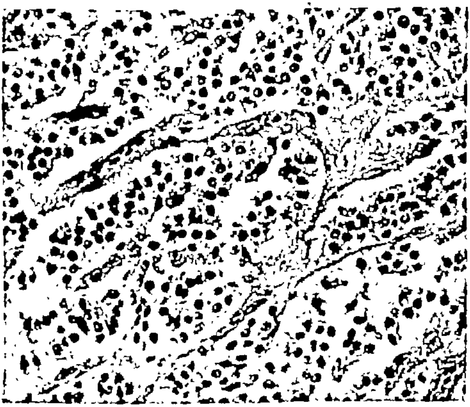

Fig. 3.—Carcinoid adenoma (×400).

epitheliomas." It would be wise to return to the evaluation of the term, and to discard it. Yet further confusion should not be introduced by further coining of new names.

jacent cells. Nuclear chromatin is hyperchromatic, rather coarse. One moderate-sized nucleolus is usually present. The stroma varies. It is sometimes dense with sweeping collagenous bundles enclosing small scattered groups of tumor cells, or it may be loose, almost myxomatous in appearance, and comprise only a minor proportion of the mass. Bone, cartilage, and fat have been reported in these tumors, but none was noted in our cases.

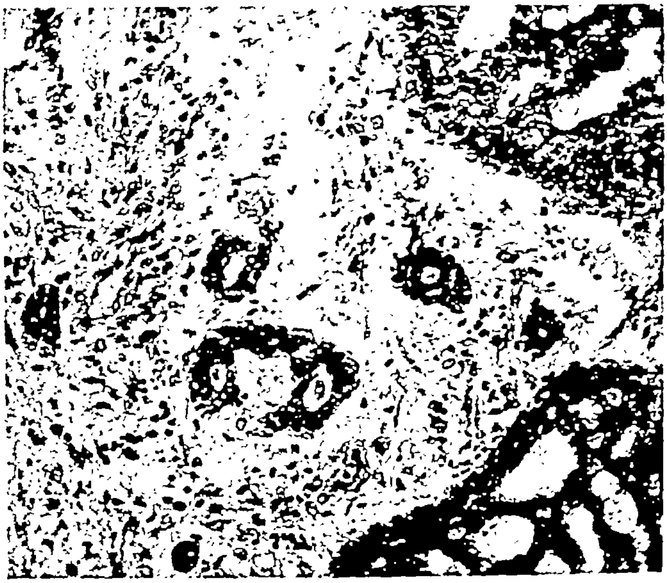

Fig. 7.—Adenocystic basal cell carcinoma of maxillary antrum (×200). Note resemblance to mixed tumor adenoma of bronchus.

Since disagreement exists as to which of these shows tendency of greater or lesser degree to metastasize, it is imperative that a generally accepted system of nomenclature be used, if the question is to be resolved. Van Hazel, Holinger, and Jensik[36] conclude that adenomas (carcinoid adenoma) are relatively benign, whereas cylindromas (mixed tumor adenoma) are malignant. On the other hand, Holly's series[18] of cases contained a number of the carcinoid type which showed distant metastasis. One of the cases here reported is that of a carcinoid adenoma with definite metastasis to the mediastinum; another had metastasized or extended, according to one's viewpoint, to nearby peribronchial nodes. Other cases have been reported of distant metastasis of carcinoid adenoma. Recently a case has been reported in which death was ascribed to the direct effects of metastases of bronchial adenoma,[25] and Cheek and Muirhead, in 1948,[41] report a case in which transition from typical bronchial adenoma to alveolar cell carcinoma pattern is described, with widespread metastasis and death. Other cases strongly suggestive of transition of adenomas to carcinomas have been reported.[8, 26] Possibly of more practical significance is the fact that both these tumors, carcinoid or mixed tumor, are known to extend or metastasize to the mediastinum, and are locally invasive. It is this feature which would seem to make surgical removal mandatory by a means which will permit adequate examination of areas to which the tumor may have spread. Mediastinal tumor will kill simply through its presence and growth whether "malignant" or not, although a patient may survive for years before this occurs. The unhappy lot of a patient with bronchiectasis, fistulae, empyema, recurrent bouts of pneumonia, and such should be kept in mind whenever survival rates are to be compared. Cases reported by Adams and others[2] illustrate the disaster invited through failure of physicians to diagnose the condition sufficiently early to permit adequate therapy.

If the tumor invades at all, then only in exceptional cases will it be possible to remove it bronchoscopically with any reasonable degree of certainty that it has been adequately removed, probably only in those with a small, easily visualized and distinct pedicle. Posterior bronchotomy is applicable, as its proponents indicate, in selected cases and has the advantage of permitting some examination of the mediastinum. In the great majority of cases we feel that surgical exploration with lobectomy or pneumonectomy and removal of mediastinal nodes, where indicated and possible, is the procedure of choice. The malignant propensities of these tumors are becoming more generally recognized as unquestionable instances of metastasis and are reported one after the other, yet one is surprised to realize how great is the lag in general acceptance of recently discovered facts in the case of a fairly uncommon lesion such as these represent. One can hardly expect, however, other professional men to develop concrete opinions regarding these tumors when they remain such a source of disagreement among thoracic surgeons themselves.

As to comparative malignancy, it appears that we are dealing with two types of tumor, possibly representing the same basic tumor, both of which are of low-grade malignancy, both of which are known to invade the mediastinum by metastasis or extension, and both, therefore, carrying similar potentialities with regard to the risk to the patient. It would seem that no purpose is served in distinguishing them at the present time so far as definitive treatment is concerned.

SUMMARY

1. Five cases of adenoma of the bronchus are presented.

2. Attention is called to the confusion resulting from the use of varying and nondescript nomenclature. It is suggested that "Adenoma" be reserved for the group as a whole, that "Carcinoid Adenoma" and "Mixed Tumor Adenoma" be used exclusively in classifying these since they are adequately descriptive terms and have been previously used. The term "Cylindroma" should be discarded.

3. It is pointed out that three, at least, of the five cases reported could not have been properly treated by bronchoscopic instrumentation alone. Nothing less than surgical exploration can offer clear evaluation of the condition and permit definitive treatment because the bronchial adenoma extends or metastasizes peribronchially and to the mediastinum.

4. In any patient suffering from repeated episodes of pulmonary inflammation, chronic cough, hemoptysis, and so on without adequate explanation, bronchial adenoma should be suspected and ruled out.

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BRONCHIAL ADENOMA IN A SUPERNUMERARY TRACHEAL LOBE

REPORT OF AN UNUSUAL CASE

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BRONCHIAL adenoma is no longer a rarity in the recent medical literature. The increasing diagnostic acumen in the recognition of the bronchial adenomas and their favorable prognosis following surgical removal has certainly been sufficient reason for the large series of case reports in the last decade.[6-8, 12-18]

The case which is to be described here is of interest because of the adenoma involving a supernumerary lobe originating from the lateral wall of the trachea slightly above the orifice of the right main stem (Fig. 5).

CASE HISTORY

W. D., No. 14,186, white male, 32 years of age, was admitted to the Veterans Administration Center Feb. 3, 1950.

Past History.—The patient became ill about ten years ago (Fig. 1), while in service—complaining of chest pains in the right side, nonproductive cough, and blood-streaked sputum. He was hospitalized on seven different occasions for the same complaint prior to entrance to our hospital. Bronchoscopy performed elsewhere in November, 1944, revealed edema and occlusion of the right upper lobe bronchus. There was no air exchange through the lumen of this bronchus. Biopsy report from that area was not conclusive, and exploratory thoracotomy was performed in March, 1945. The following was reported by the surgeon:

''The hilus was fixed and incorporated into a firm, nodular mass which was closely adherent to the pericardium, and thought to be too fixed for removal. This mass was aspirated and found to be firm and homogeneous, and there was no pus or other fluid in the two areas aspirated.''

The patient was discharged on Aug. 6, 1945, without much improvement. During the next five years he consulted several physicians without any definite result.

Physical Examination.—Physical examination on present admission revealed a fairly well-developed, slightly undernourished white man who appeared chronically ill. Temperature, pulse, and respirations were within normal limits. Examination of the chest revealed dullness to flatness on percussion with diminished to absence of breath sounds over the upper third of the right chest. There were no râles or wheezing present. The remainder of the lung fields were clear and resonant throughout. The roentgenogram (Fig. 2) revealed a density in the right upper lung field with its lower border sharply delineated at the level of the second anterior rib. Other portions of the lung were well aerated. The trachea, heart, and mediastinum were in normal position.

All the laboratory findings including sputum, as well as electrocardiograph, were not significant. Bronchoscopy showed that the opening of the right upper lobe bronchus originated slightly above the carina. Little attention was paid, however, to this abnormality at the time of the examination. In addition to the above finding, there was also marked stenosis of the upper lobe bronchus with edema and signs of chronic irritation. The biopsy material from the affected bronchus showed again only atypism of the bronchial epithelium without any definite evidence of an adenoma.

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*Chief of Thoracic Surgery, Veterans Administration Center, Temple, Texas. Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

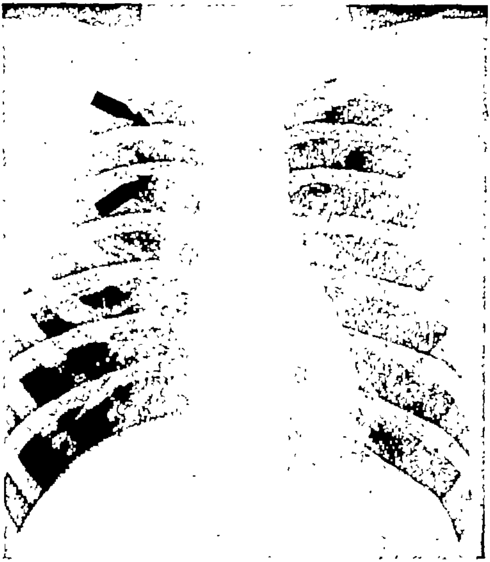

Fig. 1.—Dec. 1, 1941, roentgenogram of the chest showing abnormal density in the right upper lobe as outlined by the two arrows.

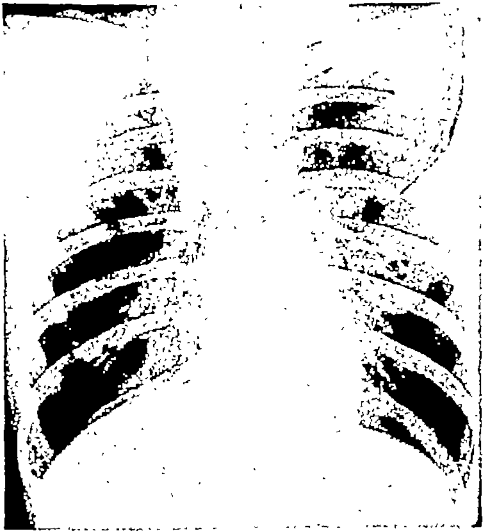

Fig. 2.—Preoperative roentgenogram showing the intrathoracic mass on Feb. 3, 1950.

Thoracotomy was performed on Feb. 14, 1950, under intratracheal gas-oxygen and ether anesthesia, through a posterolateral approach. It was found that this patient had a supernumerary lobe. The oblique fissure in this case was running anterior and the horizontal fissure was posterior to it, as one would see it in a mirror image (Fig. 3). The so-called upper lobe was occupied entirely by a growth which was attached firmly to the chest wall, cupola of the chest, and upper mediastinum. The lung was freed with great difficulty by sharp and blunt dissection from its attachments. The blood supply to the supernumerary lobe consisted of only three short ascending arterial branches from the inferior pulmonary trunk and several venous tributaries running to the superior pulmonary vein. The venous tributaries were located rather deep in the pulmonary tissue. The pulmonary ligament was attached to the anterior lobe, which suggested the possibility of a reversal of the lobes. During removal of the bronchus, it was necessary to diminish the lumen of the trachea to some extent.

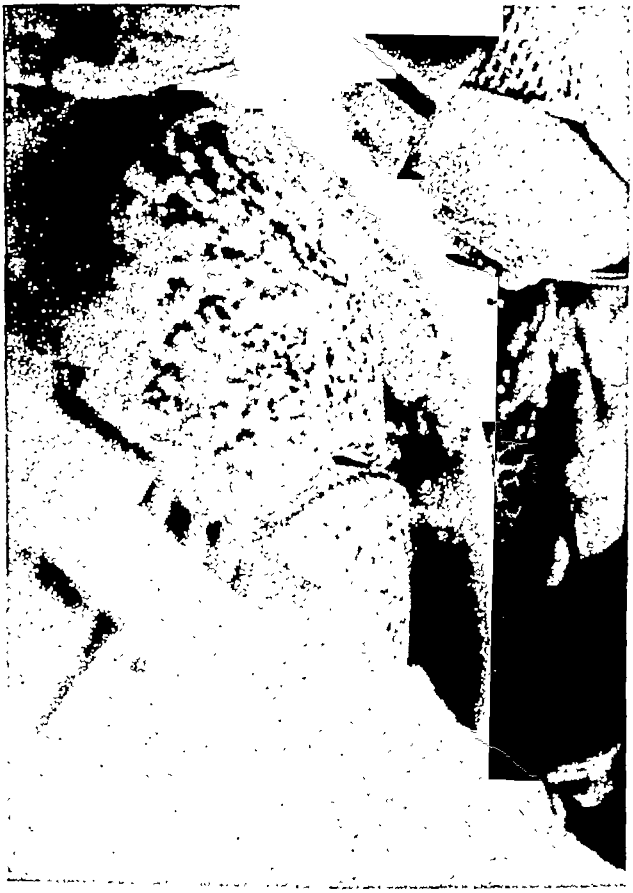

Fig. 3.—Photograph showing the horizontal fissure anteriorly and the oblique fissure posterior to it.

The patient made an uneventful recovery, and was discharged on the fourteenth postoperative day.

Pathology.—The specimen removed consisted of a tumor mass measuring 12.5 by 8.5 by 5.5 cm. (Fig. 4), the external surface of which was reddish-brown and shaggy. The pathologist reported "a partly solid and partly cystic mass with scattered areas of small bits of calcified substance. The cystic portion is multilocular, and the individual cysts vary in diameter from 5 to 20 mm., and contain thick yellowish mucoid material lined with a white membrane. The entire tumor is enclosed in a fibrous capsule about 3 mm. in thickness."

The bronchus was distorted, compressed, but not ulcerated by the tumor. The microscopic report (Fig. 5) was as follows: "The tumor is composed of small rounded cells of almost uniform size. The cells are arranged in solid nests, acinar formations, and interlacing festoons. There is present a scanty hyaline fibrous stroma. The tumor involves the wall of the large bronchus and, except for a small area of ulceration, the mucosa is intact over it. The mucosal cells are well differentiated. The capsule proves microscopically to be a *pseudo-capsule. There are many compressed bronchi with inflammation in their walls* and with patchy ulceration of the mucosa. Fibrosed collapsed pulmonary tissue is also present in the capsule with many blood vessels and nerves. Scattered solid areas resembling intestinal carcinoids are seen. No evidence of metastasis is found in the lymph node. The calcified material noted grossly was not included in the sections." The pathologic diagnosis was: Bronchial adenoma, carcinoid type.

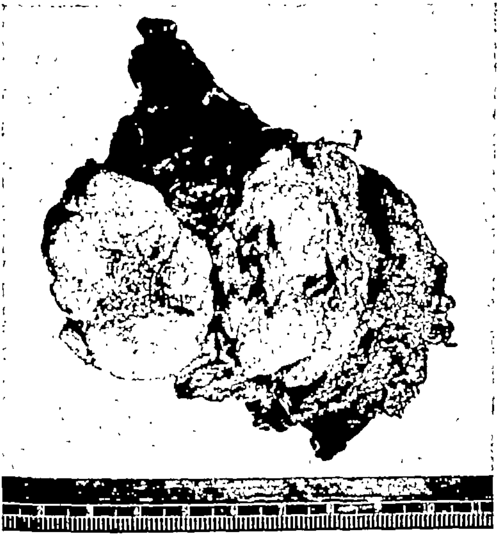

Fig. 4.—Photograph of the excised lobe showing the tumor occupying the entire supernumerary lobe. Observe the multilocular cysts with several small calcified areas in it.

Follow-up roentgenograms with lipiodol study on April 13, 1950, one month after the operation, showed a normal bronchogram of the right lung (Fig. 6). From these films, taken separately after each lobe was filled individually with lipiodol, it was found that the patient still had three separate bronchi connected with lobes on the right side, even after the removal of the so-called fourth lobe.

The patient now has no complaints and is able to follow his present occupation.

DISCUSSION

It is a well-known fact that bronchial adenomas are tumors associated with other abnormalities of the lungs. A thorough search through all pertinent literature failed to reveal a case of similar nature: an adenoma involving a

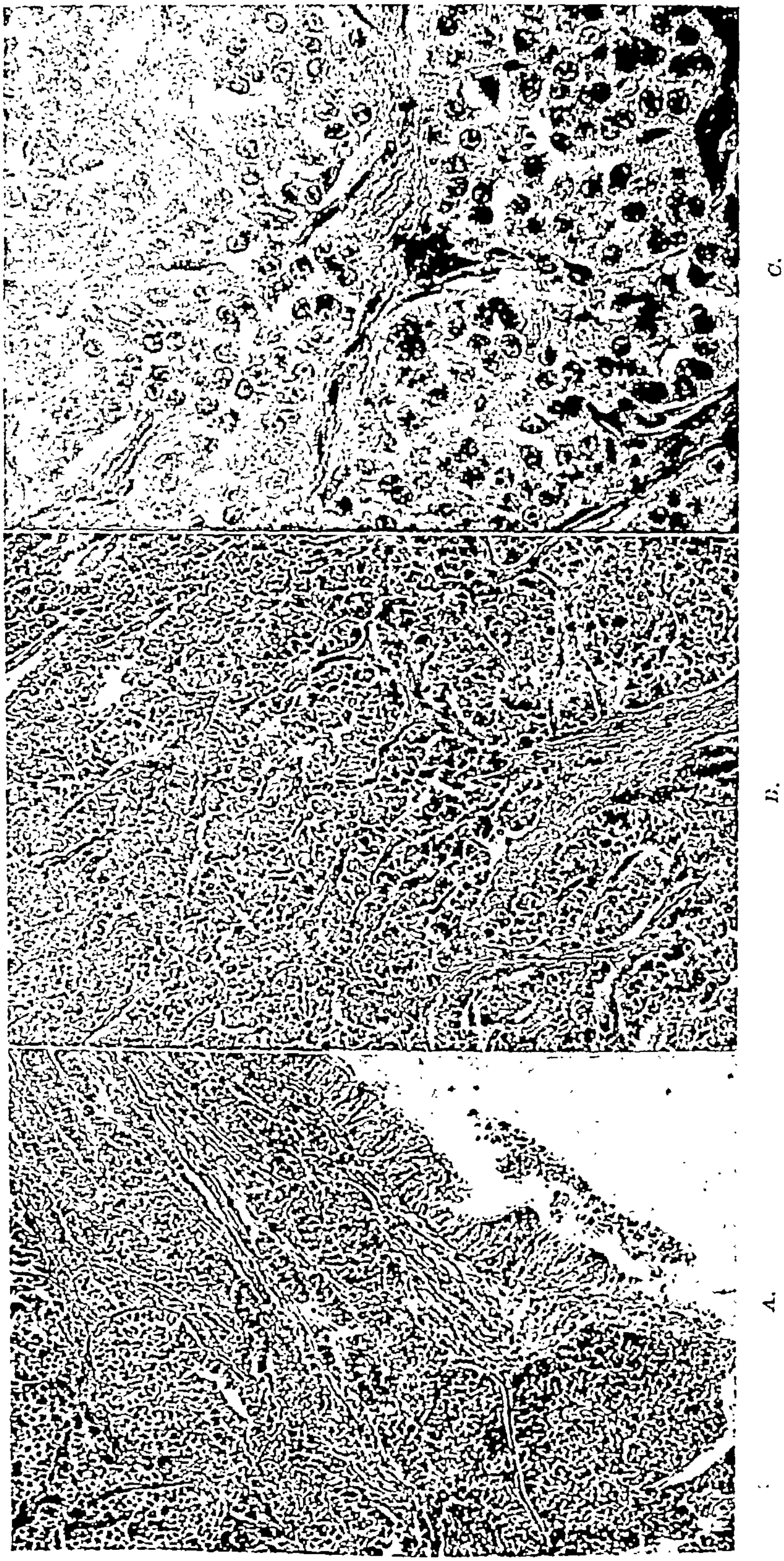

FIG. 5.—*A*, *B*, and *C*, Photomicrographs showing the histology of the tumor and its pseudocapsule.

bronchus which arose from the lower end of the trachea. Graham[10] performed a right pneumonectomy on March 7, 1935, for a mixed tumor arising from the bronchus in the right lower lobe, and during the resection he found several abnormal bronchi connecting the upper lobe directly with the trachea. Overholt,[15] on the other hand, cautions about the possibility of an accessory right upper lobe originating from the trachea which one may find during resections; but no one has formerly reported the presence of a tumor in a supernumerary lobe.

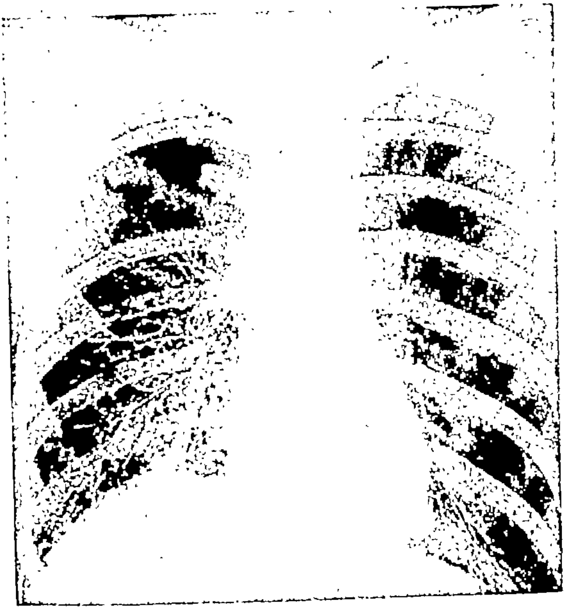

Fig. 6.—Lipiodol bronchogram which demonstrates the filling of all three lobes on the right side.

Embryologic maldevelopment has to be the probable explanation for this anomaly. Arey[4] has stated that some variations may occur in the size and the number of the major lobes of the lungs. For example, the eparterial bronchus in the human on the right side may originate directly from the trachea as in the sheep, pig, and ox; it may also imitate the relationship of the structures on the left side. Graham and Womack explain the etiology of many lung tumors as being due to failure of the embryonic bronchial buds to develop into normal structures. Although positive knowledge for the cause of developmental anomaly is lacking, there is reason to believe that in formation of the embryonic organs, most likely some intrauterine disturbance occurred.

Womack and Graham's[19] conception is that bronchial adenoma in which endodermal elements predominate are more malignant than others. It is true that the histopathology of so-called bronchial adenomas, as Graham has pointed out for many years, should be considered as potentially malignant tumors in spite of the fact that they usually remain benign for a long time. Authentic instances of metastases to the adjacent lymph nodes, vertebra, or liver have been reported by many authors.[1, 2, 3, 5, 9, 11, 19]

Since careful microscopic search through various parts of the tumor failed to show any evidence of invasiveness of the tumor into the regional lymph nodes, and since the histologic character of the specimen herein described was that of an adenoma, carcinoid type, the tumor was considered definitely as benign, and lobectomy only was performed.

SUMMARY

1. An unusual case of bronchial adenoma in a supernumerary lobe on the right side has been presented.
2. Several anomalies were associated with the tumor.
3. In spite of its severe symptomatology for almost ten years, the tumor remained benign.
4. The embryology of bronchial maldevelopment was briefly discussed.
5. Surgical extirpation is the present day accepted treatment for the bronchial adenoma.
6. The carcinoid type of bronchial adenomas is apparently more benign than the mixed type tumors of the lungs.

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TABLE X. REVISION AFTER DRAINAGE PROCEDURE

	NO. OF CASES
1. After pneumonotomy	
one revision	9
two revisions	5
three revisions	1
2. After empyema drainage	
one revision	1
two revisions	2

Brain abscess developed in seven patients who were surgically drained, in no patients in whom resection was employed. Six of the seven patients died of this complication; one patient was cured by excision of brain abscess. In two of four patients examined postmortem, undrained lung abscesses were found.

LATE RESULTS

An evaluation of the status of the patients at least one year following the latest case has been attempted. Our estimates of the final results were based on individual examinations or questionnaires. The questionnaire included inquiry regarding cough, sputum, hemoptysis, pain, and physical activity. A significant proportion of patients could not be contacted.

Of the 105 patients treated, 41, or 39 per cent, were considered cured; 31, or 30 per cent, were dead from their disease; 13, or 12 per cent, were living but not cured; and 20, or 19 per cent, could not be traced.

Patients treated by drainage:

63 cases—19 deaths (from complications of lung abscess or from operation)
    44 survivals
        11 secondary resections
            2 deaths
            4 cured
            2 improved
            3 not traced
        33 others
            6 not traced
            1 died from other disease
            26 followed

Of the 26 patients followed, 20 could be considered cured and six improved. Of the six improved patients, three still had some drainage from the wound, six continued to raise sputum, five had coughed up blood since discharge, and six had varying degrees of chest pain. Thus only 32 per cent of the original 63 patients were known to be cured without resort to secondary resection. With complete follow-up it was statistically possible for 41 per cent to have obtained a cure.

Patients treated by primary resection:

31 cases—7 deaths
    24 survivals
        8 not traced
        16 followed

Of the 16 patients followed, 13 could be considered cured, one improved, and two unimproved. Of the three patients not cured, three were still having some drainage from the wound, three continued to raise sputum, two had coughed up blood since discharge, and two had varying degrees of chest pain. Thus of the original 31 patients, 42 per cent were known to be cured. With complete follow-up, it was statistically possible for 68 per cent to have obtained a cure.

Patients treated by secondary resection (includes 11 patients previously drained at Barnes Hospital and 11 patients drained elsewhere):

22 cases—5 deaths
17 survivals
5 not traced
12 followed

Of the 12 patients followed, eight could be considered cured and four improved. Of the four patients not cured, none were having drainage from their wounds, four continued to raise sputum, four had coughed up blood since discharge, and three had varying degrees of chest pain. Thus only 36 per cent of the original 22 patients were known to be cured. With complete follow-up, it was possible for 59 per cent to have obtained a cure.

DISCUSSION

Valle[2] previously reviewed the cases of lung abscess seen on the Chest Surgical Service at Barnes Hospital between 1932 and 1943. One hundred sixty-four patients with lung abscess were treated surgically, with a mortality rate of 38.8 per cent. Twenty-eight lobectomies and two pneumonectomies were included in that series, but a breakdown of the mortality rate into drained and resected cases was not presented. The reduction in mortality to 30 per cent during the six-year period 1943 through 1948, and to 16 per cent from 1946 through 1948 is gratifying, but even these more recent figures emphasize the serious nature of the disease. Our series includes a high percentage of multiple and multilobar abscesses which would suggest a high proportion of neglected cases with complicated disease.

Comparison with other reported series is of interest. Sweet[3] analyzed the cases of lung abscess seen at the Massachusetts General Hospital between 1940 to 1945. Seventy-five patients were subjected to operation, with a mortality of 15 per cent. A cure rate of only 31 per cent was obtained in the drained patients without resort to secondary resection. Fifteen primary lobectomies and eleven secondary resections were performed with two deaths. No pneumonectomies were included in his series. The series reported by Neuhof[1] is hardly comparable since it contains only cases of putrid lung abscess drained within six weeks of the onset of the disease. However, he obtained the extremely low mortality rate of 2.3 per cent using drainage as the only method of surgical attack. In this same series he indicated a very high cure rate.

Recently several authors have reported very favorable results with the frequent use of pulmonary resection. Shaw and Paulson[4] reported 52 resections performed for chronic putrid lung abscess with two deaths, a mortality of 3.8 per cent. The postoperative empyema rate, however, reached 28.8 per cent. Kent and Ashburn[5] reported 30 cases of chronic lung abscess treated similarly with two deaths, a mortality of 6.6 per cent, with an empyema rate of 6.6.

The trend on the Chest Surgical Service at Barnes Hospital in the past few years has led to the more frequent use of lobectomy and pneumonectomy. Segmental resection has been employed only rarely. As specific indications for the employment of primary resection in the treatment of lung abscess, the following may be listed: (1) multiple lung abscesses; (2) associated bronchial stenosis; (3) extended chronicity with fibrosis and secondary bronchiectasis; (4) suspicion of bronchial adenoma or carcinoma; (5) suspicion of infected congenital lung cyst; (6) abscess somewhat inaccessible to drainage; (7) simple lung abscess in the good risk patient which is failing to respond to conservative treatment.

SUMMARY

1. As a result of improved surgical techniques and extensive use of antibiotics, the mortality of surgically treated lung abscess at the Barnes Hospital has declined from a previously reported rate of 38.8 per cent prior to 1943 to 16 per cent from 1946 through 1948.

2. The frequent employment of pulmonary resection in many instances in preference to drainage has contributed significantly to reduction in mortality as well as improvement in the cure rate, particularly in the complicated cases.

3. The postresection empyema rate decreased from 68 per cent from 1943 through 1945 to 21 per cent from 1946 through 1948. This reduction coincided with intensive use of antibiotics as well as the more general application of individual ligation technique.

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BRONCHOSCOPY IN THE TREATMENT OF SPONTANEOUS AND TRAUMATIC PNEUMOTHORAX

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THE treatment of spontaneous or uncomplicated traumatic pneumothorax, as a rule, presents few difficulties. The air in the pleural cavity is absorbed in a few weeks; at most, one or more aspirations are necessary to hasten re-expansion of the lung. In the presence of tension pneumothorax associated with intrathoracic pressure symptoms, continuous air deflation is instituted by means of water-sealed drainage with or without negative suction. The instillation of irritating substances into the pleural cavity in an attempt to induce a sterile pleurisy and thereby obliterate the pneumothorax is another method of treatment. Silver nitrate, hypertonic glucose solution, gomenol, or the patient's own blood is used for the purpose. But inasmuch as the induction of so-called provocative or chemical pleuritides is not always effective nor devoid of danger of causing an intense pleural reaction with possible permanent impairment of lung function, this method of treatment is losing favor. Instead, an increasing number of patients with chronic or recurrent pneumothorax are being submitted to operation with the aim of resecting the ruptured bleb or cyst, or even the entire lobe if the latter is a source of repeated blowouts. Direct surgical treatmet is also displacing intrapleural pneumonolysis which is used occasionally to free the lung of suspending adhesions permitting closure of the pleural opening. The forementioned are, in brief, the current methods of treating nontuberculous pneumothorax.

Except for passing reference to the use of bronchoscopy for diagnostic purposes, a review of the literature on spontaneous and traumatic pneumothorax does not reveal a single report which stresses the need of this measure as an integral part of the treatment in certain cases. This is surprising in view of the fact that a lung collapsed by pneumothorax may fail to reinflate not because of factors relating to the lung rupture or to the pneumothorax, but as a result of bronchial obstruction. Viscid secretion, clotted blood, or other types of impaction within bronchi or bronchioli may prevent the ingress of air into the lung, thus maintaining the organ in a state of collapse. This has been our experience in two patients with spontaneous pneumothorax and two with traumatic pneumothorax. In each instance bronchoscopy, when other measures failed, was used with gratifying results.

REPORT OF CASES

CASE 1.—T. K., a student, aged 17 years, developed an upper respiratory infection. That same night he was seized with severe chest pain, fever, cough, and dyspnea. He was admitted to a hospital where he was given penicillin for suspected pneumonia. A roentgeno-

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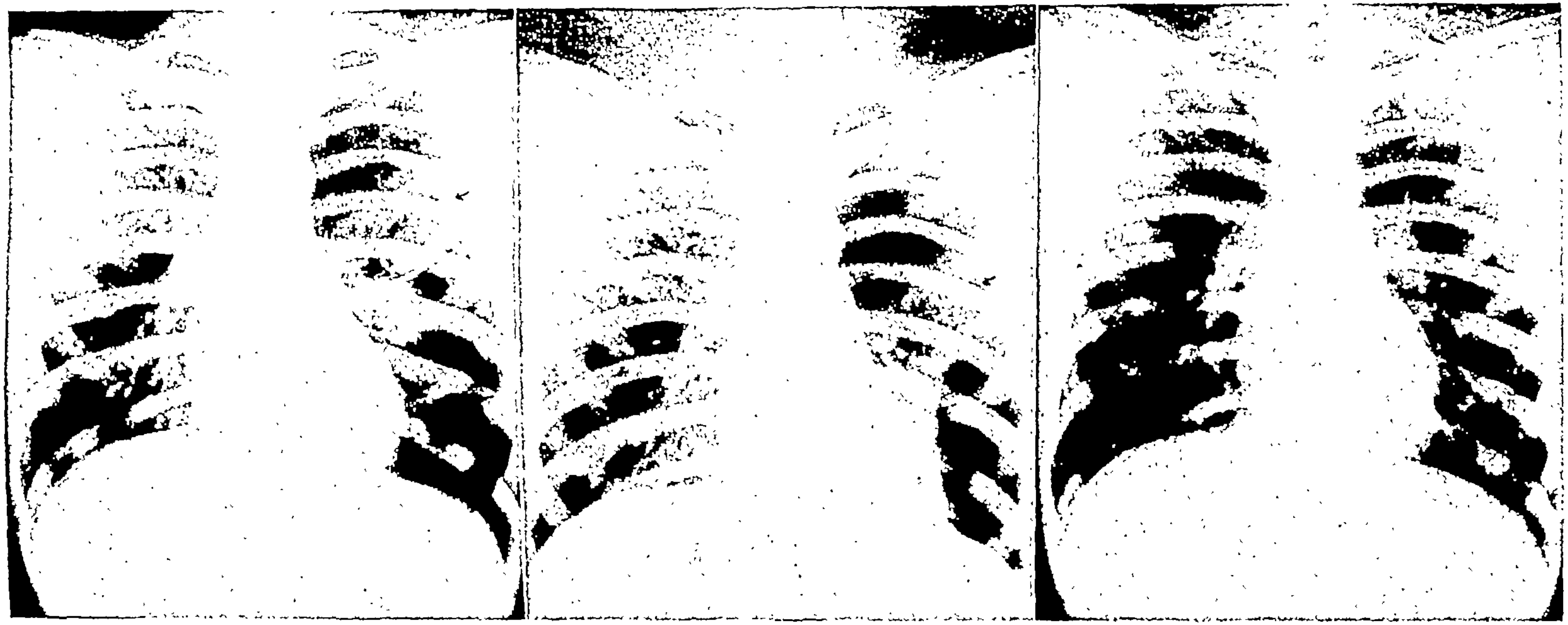
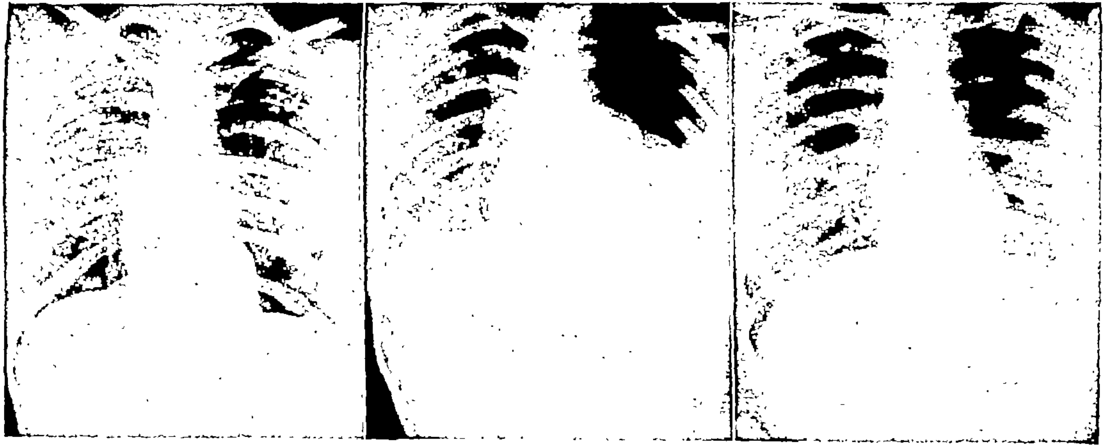


A. *B.* *C.*

Fig. 1.—(Case 1, T. K.) Roentgenograms of a boy of 17 years showing atelectasis in the presence of a spontaneous pneumothorax. Before and after bronchoscopic aspiration. *A*, Feb. 24, 1950. Pneumothorax on left collapsing about 50 per cent of lung; no adhesions or fluid; heart not displaced. *B*, Feb. 28, 1950. Increase in size of pneumothorax the left lower lobe being almost completely collapsed; small amount of fluid at left base; heart slightly displaced to left. (Bronchoscopy, March 7, 1950.) *C*, March 20, 1950. Pneumothorax completely absorbed; no gross abnormalities seen.



A. B. C.

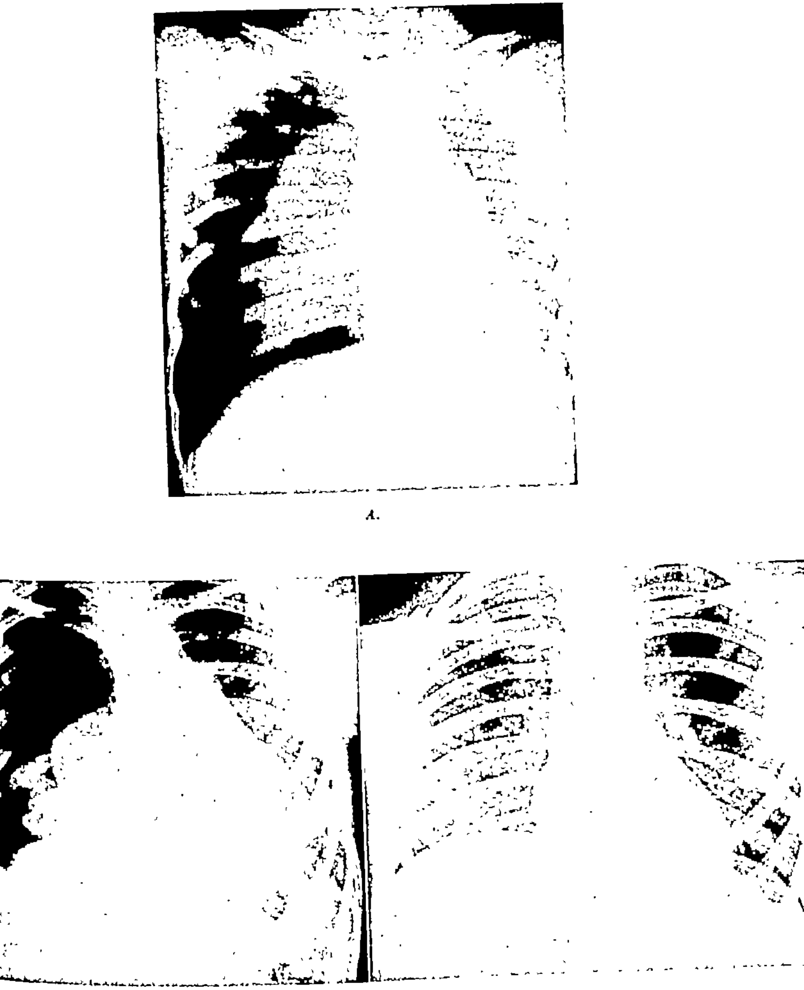
Fig. 2.—(Case 2, T. S.) Roentgenograms of a woman of 20 years showing atelectasis and massive effusion in the presence of a spontaneous pneumothorax. Before and after bronchoscopic aspiration. *A*, March 8, 1949. Pneumothorax on *left* collapsing about a third of the lung; no adhesions or fluid; heart slightly displaced to right. *B*, March 12, 1949. Left lung is almost completely collapsed by a massive *hydropneumothorax*. (Bronchoscopy, March 12, 1949.) *C*, April 30, 1949. Left lung has reexpanded; a small collection of air is still seen at extreme apex.

gram of the chest the following day revealed a left-sided pneumothorax, the lung being collapsed to about 50 per cent of its volume. The heatrt was not displaced; no fluid or adhesions (Fig. 1*A*). Aspiration of air from the chest was followed by slight relief. Four days later, a roentgenogram revealed a decided increase in the degree of pulmonary collapse, especially of the left lower lobe. A small effusion was present at the base (Fig. 1*B*). The patient's dyspnea, cough, and fever became aggravated. Repeated aspirations of air did not reduce the size of the pneumothorax. At this time the patient was seen by us. It was suspected that he had a bronchial obstruction due to impacted sputum incident to his antecedent respiratory infection, and that this was the cause of the complicating atelectasis of the left lower lobe. Bronchoscopic aspiration was followed immediately by partial aeration of the lung with coincident improvement in the patient's symptoms. By the end of a week, and without the need of additional air removal, the lung reexpanded about 50 per cent and in three weeks, completely. Neither lung revealed parenchymal disease (Fig. 1*C*).

CASE 2.—T. S., housewife, aged 20 years, became suddenly ill with severe pain in the left chest, shoulder, and arm. After twenty-four hours, she developed increasing shortness of breath so that by the time she consulted us she was in acute respiratory distress. A roentgenogram of the chest taken the day after the seizure showed a pneumothorax collapsing about a third of the left lung. There was no fluid in the pleural space, no adhesions, and only slight displacement of the heart to the right (Fig. 2*A*). However, a film taken at the time she came to our office four days after the seizure revealed almost complete collapse of the left lung with fluid occupying half of the pleural cavity (Fig. 2*B*). During the following two days, three aspirations were made of serous fluid (totalling 1,000 c.c.) and an indeterminate amount of air. The intrapleural pressure which was initially slightly positive became negative. Because of the persistent cough and expectoration of thick green sputum, which the patient had had from the onset of the illness, as well as the sudden marked change in the appearance of the roentgenogram, it was suspected that bronchial obstruction was a complicating factor. Bronchoscopy revealed the right main bronchus and all its subdivisions normal and free of secretions. The left main bronchus appeared narrowed; the branch to the lower lobe contained thick green secretion which was aspirated. The following day the patient felt greatly relieved. A roentgenogram taken two days later showed considerable reaeration of the left lung. In six weeks, and without additional aspirations, the pneumothorax was absorbed, except for a small residue of air at the extreme apex (Fig. 2*C*). A subsequent film showed the lung completely reexpanded.

CASE 3.—N. M., a girl, aged 8 years, was brought by ambulance to the Morrisania City Hospital after having been struck by an automobile. The child was in critical condition, unconscious, cyanotic, and dyspneic. She was placed in oxygen, an infusion of 500 c.c. of plasma started, given penicillin and other supportive measures. A roentgenogram of the chest taken the day of the accident revealed a partial collapse of the right lung by a pneumothorax with displacement of the mediastinum and heart into the left chest. A fracture was noted of the right eighth rib posteriorly (Fig. 3*A*). A roentgenogram the following day showed a marked increase in the degree of collapse of the lung, especially of the right upper lobe; fluid was present at the base (Fig. 3*B*). It was suspected that a superimposed atelectasis had occurred as a result of bronchial obstruction. Two chest aspirations yielded about 200 c.c. of air and 180 c.c. of serosanguineous fluid, but the lung failed to reexpand. Bronchoscopy was done the following day and viscid sputum aspirated. The patient's condition took an immediate turn for the better. The air in the chest was quickly absorbed, without the need of further aspirations, and by the time of discharge, two weeks later, the lung had reexpanded completely (Fig. 3*C*).

CASE 4.—V. K., a man, aged 22 years, was admitted to the Morrisania City Hospital three and one-half hours after having been stabbed twice in the right axilla. The patient's only complaint was pain on deep breathing. Examination of the chest showed a closed stab wound in the right lower chest. The breath sounds were diminished to absent on that side. A roentgenogram taken the same day revealed a right-sided pneumothorax with the lung



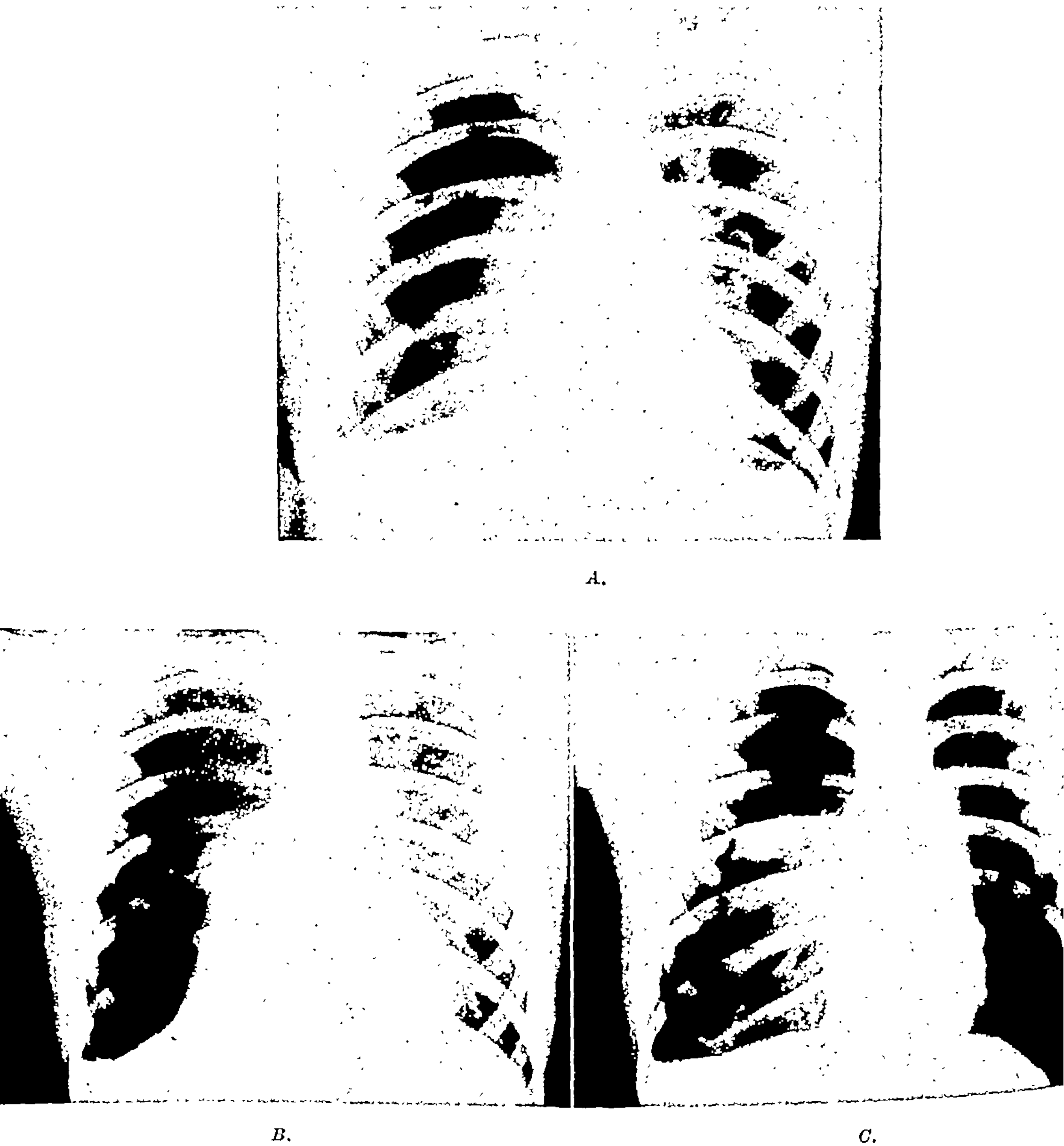
A.

B.

C.

Fig. 3.—(Case 3, N. M.) Roentgenograms of a girl of 8 years showing atelectasis in the presence of a traumatic pneumothorax. Before and after bronchoscopic aspiration. *A*, June 8, 1949. Pneumothorax on right collapsing about half of lung; no adhesions or fluid; considerable displacement of heart and mediastinum to left; fracture of eighth rib on right. *B*, June 9, 1949. Marked increase in degree of collapse of right lung, especially of upper lobe; small amount of fluid at right base (Bronchoscopy, June 10, 1949). *C*, June 24, 1949. Right lung completely reexpanded; no gross abnormalities seen.

collapsed about 50 per cent of its volume. There were no adhesions; a small effusion obliterated the right costophrenic sinus. The mediastinal contents were not displaced (Fig. 4*A*). The treatment consisted of air removal and intrapleural instillations of penicillin and streptomycin. Eleven days later, a comparative film showed a marked increase in the size of the pneumothorax with complete collapse of the right lung (Fig. 4*B*). At this time, the patient developed dyspnea. On the assumption that the right lung had sustained a superimposed atelectasis, bronchoscopy was done, and a large amount of mucus aspirated. Following bronchoscopy, the patient experienced immediate relief and no longer required air



A.

B. *C.*

Fig. 4.—(Case 4, V. K.) Roentgenograms of a man of 22 years showing atelectasis in the presence of a traumatic pneumothorax. Before and after bronchoscopic aspiration. *A*, June 22, 1950. Pneumothorax on right collapsing about 50 per cent of lung; no adhesions; small amount of fluid at right base. Subcutaneous emphysema in right axillary region. *B*, July 3, 1950. Marked increase in size of pneumothorax, the right lung almost completely collapsed. (Bronchoscopy, July 7, 1950.) *C*, July 10, 1950. There is considerable reexpansion of right lung; a patch of atelectasis still present in right upper lobe; small effusion persists.

aspirations. A film taken three days later showed considerable reaeration of the lung, a patch of atelectasis still being present in the right upper lobe (Fig. 4C). The patient left the hospital before the lung had reexpanded completely.

Our initial experience with bronchoscopic suction in the treatment of a nonexpanding lung was in a dentist, aged 37 years, who developed a spontaneous pneumothorax immediately following an injection of histamine which he had been receiving for the relief of migraine. The patient was taken to a hospital where a pneumothorax was found collapsing about 75 per cent of the right lung. After several days, he returned home, comfortable but somewhat dyspneic. Three weeks later, shortly before consulting us, he had a recurrence of chest pain and the dyspnea became severe. Examination at this time revealed a right-sided pneumothorax with almost complete collapse of the lung. For several days the patient was treated by daily withdrawals of 500 to 1,000 c.c. of air. It was noted that after the removal of air the pressure, which was initially positive, became negative, but as soon as the patient began to cough and exert himself the pressure became again positive. This, and the fact that several of the films revealed a circumscribed translucency overlying the right upper lobe, suggested that a ruptured bleb was the underlying cause. In the belief that bronchial obstruction, incident to a probable bronchospasm following the injection of histamine, might be preventing reaeration of the lung, bronchoscopy was done. The right main bronchus was found occluded by a plug of gray mucus which was aspirated. The bronchi were visualized and no pathologic changes noted. In spite of bronchoscopic suction and strenuous attempts to reexpand the lung, there was little change in the patient's condition. Bronchoscopy was repeated and, at this time, an effort was made to aspirate the small bronchi by means of a narrow rubber catheter attached to the end of a metal aspirating tube. Additional measures consisted of under-water drainage and, on several occasions, the instillation of the patient's own blood into the pleura in the hope of provoking a pleural reaction. These measures were also of no avail. One day following an intrapleural injection of 100,000 units of penicillin, which the patient had received twice previously, he developed a generalized erythematous eruption of the skin associated with itching and a rise in temperature. Coincidentally, a small effusion appeared in the pneumothorax space. Shortly thereafter the pneumothorax began to be obliterated, and in two weeks the lung reexpanded completely. Although not comparable with the first four cases cited, because of complicating factors, the indication in this case for attempting to open up bronchial channels seemed clear cut. Currently, the demonstration of a bleb or cyst in a patient with chronic or recurrent pneumothorax would be an indication for exploratory thoracotomy.

DISCUSSION

In each of the first four cases cited, a partially deflated lung, in the presence of a pneumothorax, sustained additional collapse as a result of superimposed atelectasis. The occurrence of the latter was suspected from a sudden change in the appearance of comparative roentgenograms which revealed an increase not only in the extent of the collapse but also in the degree of airlessness of the affected lung. A pleural effusion often appeared coincidentally. The symptoms were in keeping, the cough, expectoration, dyspnea, and fever becoming more pronounced. Furthermore, in each case there was an antecedent history either of respiratory infection or chest trauma, predisposing factors in the development of bronchial obstruction. Inasmuch as the condition was rapidly resolved following bronchoscopy without additional air withdrawals, it would seem to confirm the diagnosis and the rationale of the treatment.

Even if bronchoscopy does not reveal obstruction, it does not necessarily exclude its presence beyond bronchoscopic view. If there is demonstrable pulmonary atelectasis, or if there is an antecedent history to make one suspect bronchial obstruction, an attempt should be made to cleanse the bronchi of secretion in any event. It is well to bear in mind that in the presence of negative intrathoracic pressure, which exists in a low-tension pneumothorax, the inspiratory pull preceding cough and exertional dyspnea serves to draw mucus from larger into smaller bronchi, and as the latter become plugged there is progressive atelectasis of distal segments of the lung. The main bronchi in such cases may not be visibly obstructed.

CONCLUSION

Bronchial obstruction with or without demonstrable atelectasis may complicate a spontaneous or traumatic pneumothorax preventing reaeration of the lung in spite of strenuous attempts to obliterate the pneumothorax space. It is essential, therefore, that in obstinate cases bronchoscopic aspiration be employed to cleanse the bronchi of secretion and allow ingress of air in addition to attempts at obliterating the pneumothorax space by conventional methods of treatment.

THE PULMONARY ARTERY IN BRONCHIECTASIS

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MUCH has been written about bronchiectasis as a disease entity, and more recently numerous investigators have called attention to the presence of bronchiectasis complicating pulmonary tuberculosis. Dormer and his associates[1-5] in South Africa, who have done bronchograms on more than 2,000 patients with tuberculosis, have suggested that the complication of bronchiectasis is very common and often is of such clinical moment as to overshadow the tuberculosis and render collapse ineffective. Norris and Landis[6] stated that bronchiectasis is present in more than 21 per cent of tuberculous lungs seen at autopsy. Jones[7] and her co-workers recently have reported on the relationship between tuberculosis and bronchiectasis, but made no mention of the possible functional differences between the disease entity bronchiectasis and bronchial ectasia encountered in tuberculosis. Liebow and associates[8] have drawn attention to the role of the bronchial arteries in this disease. The term bronchiectasis as used in this paper denotes the pulmonary disorder characterized by dilatation of the bronchi and pathologic changes in the bronchial wall and adjoining tissues which are found as a sequel to many varied and apparently unrelated conditions which include bronchopneumonia, lung abscess, neoplasm, and influenza. It does not include bronchial ectasia commonly associated with pulmonary tuberculosis. Although the study of the pulmonary arteries by injection of a contrast media is not new, little has been written about the pulmonary arteries in bronchiectasis and their possible role in altered function. The following studies were limited to the bronchial tree and to the pulmonary arteries, for they are readily accessible, and any alteration in either of these two should be reflected in an alteration in function.

MATERIALS AND METHODS

While numerous materials were used as contrast media, iodized oil was found to be most suitable. As soon as each lung specimen was obtained from either the operating room or the autopsy table, the pulmonary arteries were cannulated and the iodized oil injected under fluoroscopic observation. The cannulae were then removed, the vessels ligated to prevent retrograde spilling of the iodized oil, and roentgenograms taken. The bronchi then were injected in a similar manner with care being taken to avoid overfilling so as to prevent

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**Principal Thoracic Surgeon, Ray Brook State Tuberculosis Hospital, Ray Brook passage of the oil into the alveoli. Roentgenograms were again taken. Using this method, lungs were studied which had the classical lesions of primary bronchiectasis and tuberculosis. Normal lungs were also studied.

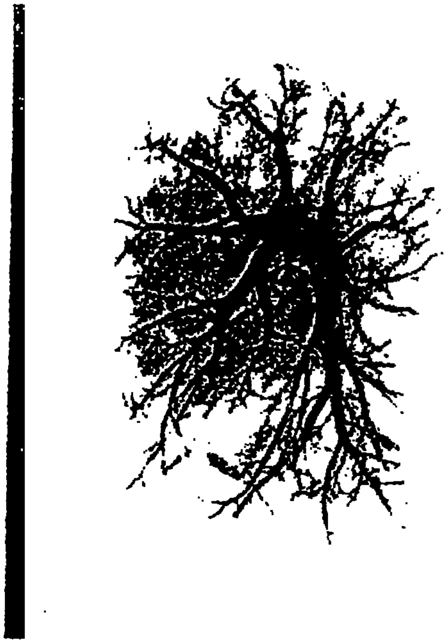

Fig. 1.—Normal lung in which the pulmonary artery has been injected with iodized oil. Note the gradual tapering and increased branching of the vessels which form a delicate and uniform pattern throughout the lung.

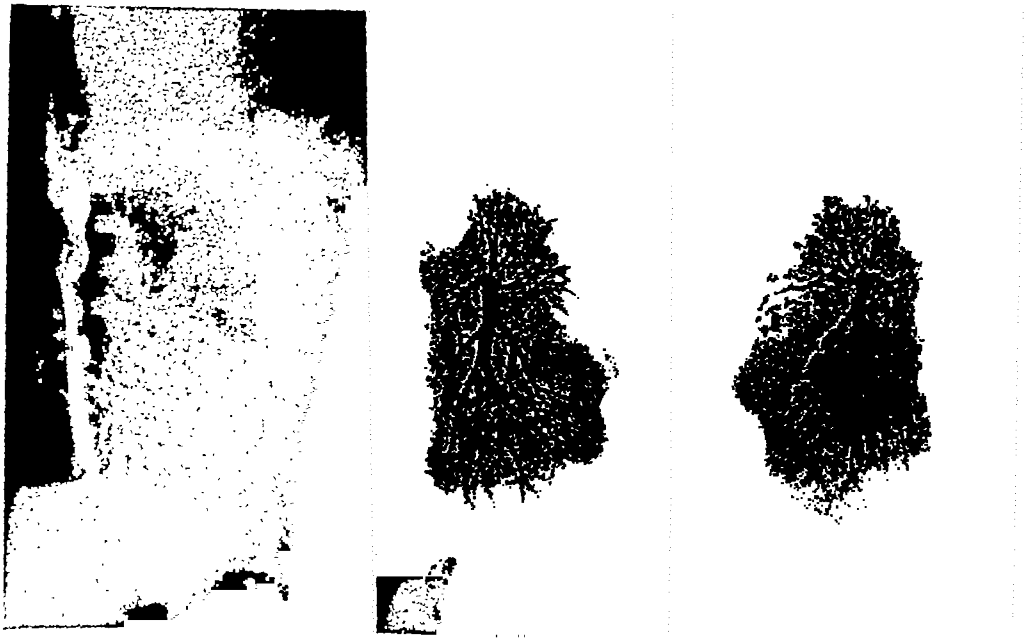

Fig. 2.—*Left.* Bronchogram of a patient with extensive bronchiectasis of the left lung. The radiolucent area in the left upper lung field represents an area of lung hernia from the right. *Middle.* Excised left lung from same patient with the pulmonary artery injected with iodized oil. Note the similarity of the pulmonary arterial pattern with that of Fig. 1. *Right.* Same lung with bronchi injected with iodized oil illustrating the extreme degree of bronchiectasis. The dense area at the periphery of the lower lobe which is not penetrated by either pulmonary artery or bronchi is due to a subpleural hematoma which developed at the *time of* resection.

RESULTS

The pulmonary artery in the normal lungs showed the major branches giving rise to smaller radicles which diminished in caliber as they approached the periphery of the lung. The gradual tapering and increased branching of the vessels formed a delicate and uniform pattern throughout the lung down to vessels of precapillary size. In no place were they widened, shortened, or distorted (Fig. 1).

The pattern of the pulmonary artery in bronchiectasis of the nontuberculous lung was the same as that seen in the uninvolved lungs (Fig. 2). The major branches were uniform in size and gave rise to small radicles which had a similar delicate arborization as observed in the normal lung.

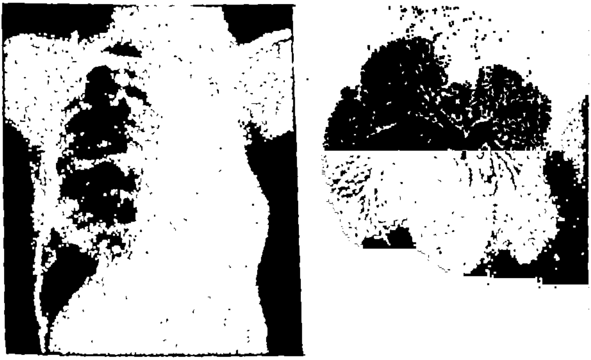

Fig. 3.—*Left.* Posterior-anterior roentgenogram of the chest taken shortly before death. This patient had fibrocaseous pulmonary tuberculosis of the entire left lung. Following thoracoplasty on the left there was spread of disease to the right lung with production of fibrocaseous disease and cavitation throughout the upper half of the lung field. *Right.* Specimen from the same case from which the heart and both lungs were removed en bloc. Note the complete absence of branches of the pulmonary artery throughout the periphery of the left lung, right upper lobe and superior segment of the right lower lobe. In the right lower lobe which is relatively free of disease there is a normal pulmonary arterial pattern.

On the contrary, in tuberculosis the pulmonary arterial pattern was markedly altered in the areas of fibrocaseous disease (Fig. 3). Many of the larger branches were shortened, narrowed, and irregularly distributed. There was a marked paucity of the smaller branches in areas of fibrotic disease, and complete absence in areas of caseation. This same pulmonary arterial pattern was present whether bronchial ectasia was present or absent in these tuberculous areas.

DISCUSSION

In a consideration of functional capacity of the lung, the presence of pathologic changes becomes important. Fibrocaseous tuberculosis is accompanied invariably by an obliterative arteritis. Thus, the areas of the lung which are poorly ventilated due to a tuberculous process are in turn poorly circulated, and one would not expect unsaturated blood returned to the left auricle from these areas. This is borne out in respiratory physiologic studies in tuberculous patients; namely, arterial oxygen unsaturation usually is not found at rest. Carvalho[9] recently found that angiocardiopneumographic examinations performed on living tuberculous patients showed the pulmonary circulation in the areas of disease to be either absent or deficient. He also states that, contrary to the above, angiocardiopneumographic examinations show increased blood supply at the region of lung abscesses. He concludes that he is thereby able to distinguish between the images of tuberculosis and those of common inflammatory nature. Steinberg[10] verified by angiocardiopneumography the avascularity of tuberculous parenchymal lesions reported by Carvalho.

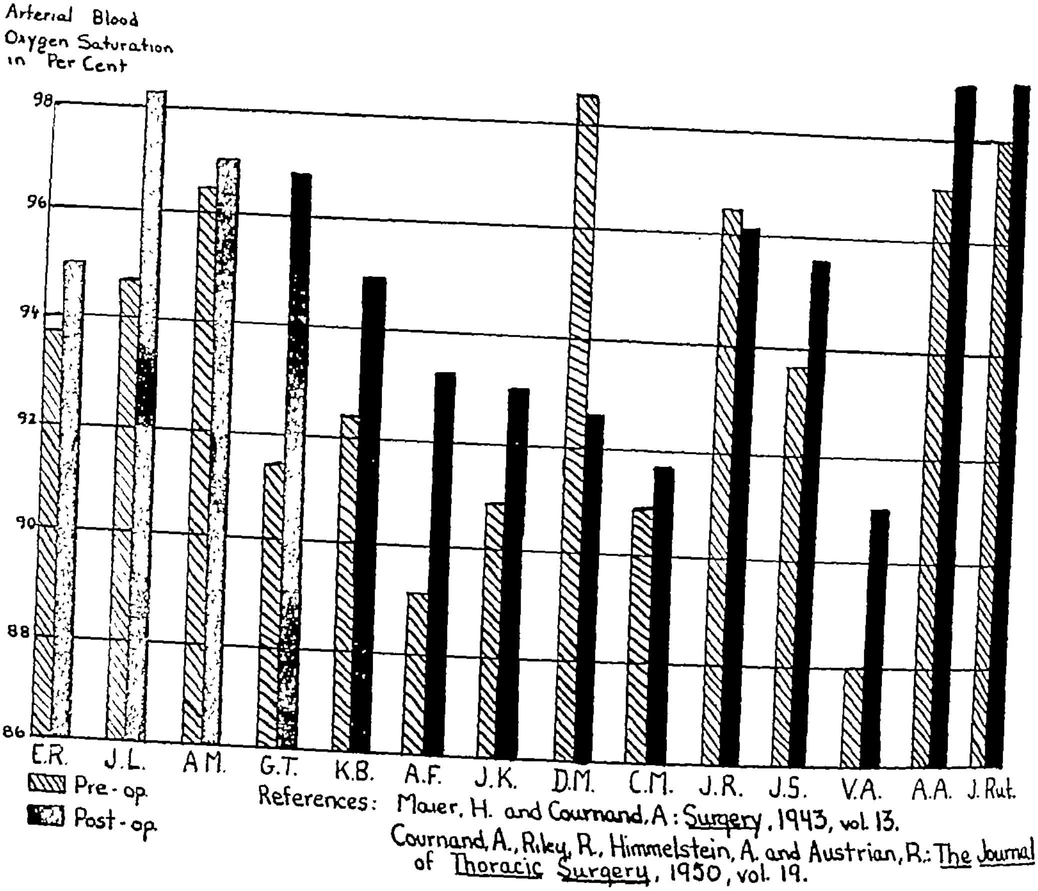

Fig. 4.—Table compiled from published papers of Maier, H., Cournand, A., Riley, R., Himmelstein, A., and Austrian, R. The first column in each group of two denotes the preoperative arterial blood oxygen saturation in per cent. The second column in each group denotes the postoperative level. There is a consistent elevation in the postoperative levels except in one case, J. R., which shows practically no change and in D. M., who shows a marked decrease in the postoperative level. For discussion see text.

Lisa and Rosenblatt,[11] in their histologic study of 110 cases of bronchiectasis, found that the blood vessels in the vicinity of the affected bronchi showed no general changes except in one case which showed a productive endarteritis. If there is blood flow through the intact pulmonary arteries that have been demonstrated above in areas of nontuberculous bronchiectasis, then there is circulation without ventilation, and one should expect decreased systemic arterial oxygen saturation. Although the blood flow through the pulmonary arteries in areas of primary bronchiectasis has not been studied, the arteries are intact for perfusion. Such an arrangement as is here demonstrated suggests a shunt mechanism in that venous blood flows through the unventilated diseased portion of the lung and returns to the left auricle as venous blood where it mixes with blood from other areas of the lung that are normally circulated and ventilated. Maier and Cournand,[12] and Cournand, Riley, Himmelstein, and Austrian[13] studied pre- and postoperative arterial oxygen saturations on a number of bronchiectatic patients who underwent pulmonary resections. They found definite improvement in the postoperative arterial oxygen saturation in twelve of fourteen cases, one patient showing no difference (Fig. 4). The patient who showed considerable decrease in his postoperative arterial oxygen saturation had the complication of postoperative left upper lobe atelectasis. Maier and Cournand,[12] in speaking of the postoperative complications of atelectasis, state that: "Since a considerable amount of blood may be flowing through the pulmonary vessels of a poorly ventilated lobe, arterial oxygen unsaturation results."

SUMMARY

Injections of the pulmonary arteries with a radio-opaque material in cases of nontuberculous bronchiectasis reveal a normal pattern in contrast to tuberculosis, and suggest that the arteries are intact for perfusion. Following resection of the bronchiectatic portion of the lung, there is an increase in the arterial oxygen saturation. This improvement is thought to be due to the removal of the poorly ventilated but adequately circulated diseased portion of the lung which is acting as a venous-arterial shunt mechanism. Respiratory physiologic studies support this observation.

In fibrocaseous pulmonary tuberculosis there is an irregularity of the large branches and a marked paucity of the small branches in fibrotic areas and complete absence in areas of caseation. The absence of circulation in the poorly ventilated areas is an explanation for the findings of normal arterial oxygen saturation in pulmonary tuberculosis at rest despite loss of considerable pulmonary tissue.

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LIVER HERNIATION SIMULATING INTRATHORACIC TUMOR

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HERNIATION of all or part of the right lobe of the liver through the diaphragm is uncommon, having been described in only six instances.[1-6] In 857 cases of diaphragmatic hernia reviewed by Hedblom,[7] protrusion of the liver into the chest occurred in only fourteen patients. Since the presence of liver tissue in the chest may closely simulate a mediastinal or pulmonary tumor, the correct diagnosis is usually not made (or entertained) prior to operation or autopsy. In order to direct attention to hepatic herniation as a possibility to be considered in the differential diagnosis of rounded shadows in the right lower lung field which are contiguous with the diaphragm, an illustrative case is cited.

CASE REPORT

A 63-year-old German sheet metal worker (N.Y.H. 507 327) was admitted complaining of epigastric pain of twelve years' duration and a twenty-five pound weight loss during the preceding year. Forty-four years ago, at the age of 19 years, he had fallen seventy-five feet, sustaining a cerebral concussion and multiple fractures; no history of abdominal distress could be obtained. Twelve years ago he first noted sharp, localized epigastric pain usually occurring about two hours after meals and relieved by belching. The pain gradually changed to a constant, dull, epigastric aching with occasional episodes of sharp, knifelike pain lasting a few minutes. There was no history of nausea, vomiting, hematemesis, jaundice, diarrhea, anorexia, bloody or tarry stools. Only a slight cough had been noted. Physical examination was essentially normal.

Roentgenogram of the chest (Fig. 1) showed a large, homogeneous, sharply circumscribed mass in the right anterior chest. This did not pulsate on fluoroscopy, nor was there any evidence of a fluid level or areas of calcification. It could not be separated from the diaphragm. On the basis of the roentgenogram appearance, the mass was thought to be a pulmonary or mediastinal tumor. Pertinent laboratory examinations were unrevealing, and exploratory operation was decided upon.

At operation a large, round herniation of part of the right lobe of the liver was encountered, passing through the central portion of the dome of the diaphragm. The herniated portion was connected to the remainder of the liver by an 8-centimeter pedicle circumscribed by a tenuous band. The entire mass was removed by transecting its base, and the resulting defect in the diaphragm closed. The patient's postoperative course was uneventful with relief of symptoms. He died one year later due to a pneumococcus meningitis.

SUMMARY

This case is unique in that the traumatic diaphragmatic hernia probably had its onset forty-four years ago. Pain referable to the lesion came on thirty-two years later and may have coincided with incarceration of the herniated

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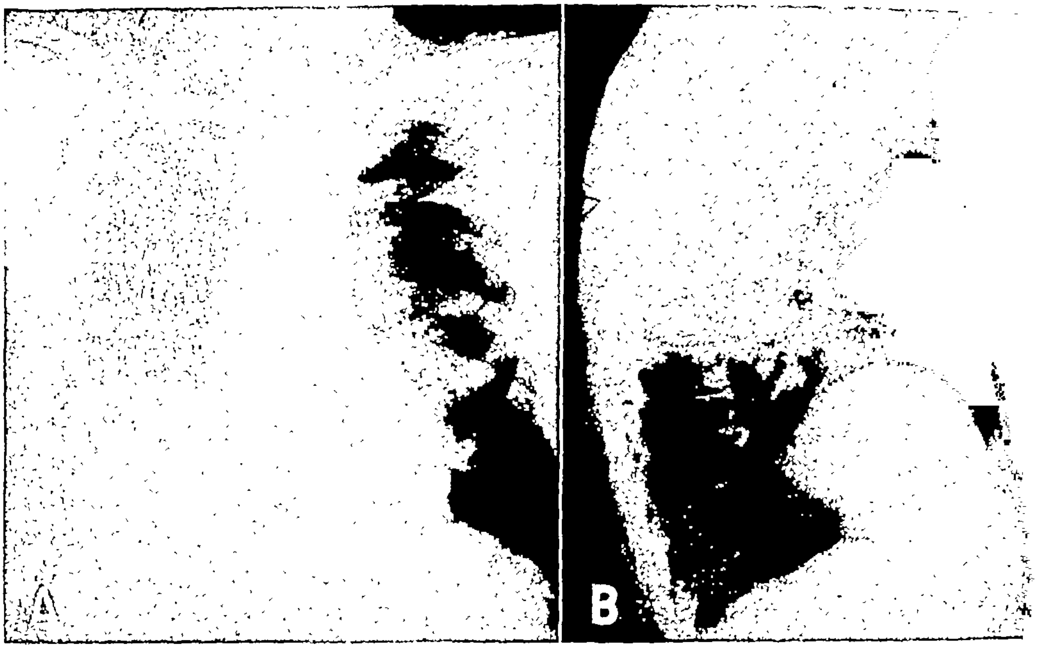

Fig. 1.—Liver Hernia. *A*, Frontal roentgenogram shows a large, rounded mass adjacent to the right heart border and contiguous with the diaphragm. *B*, Right lateral roentgenogram shows the anterior position of the mass which simulates an intrathoracic tumor.

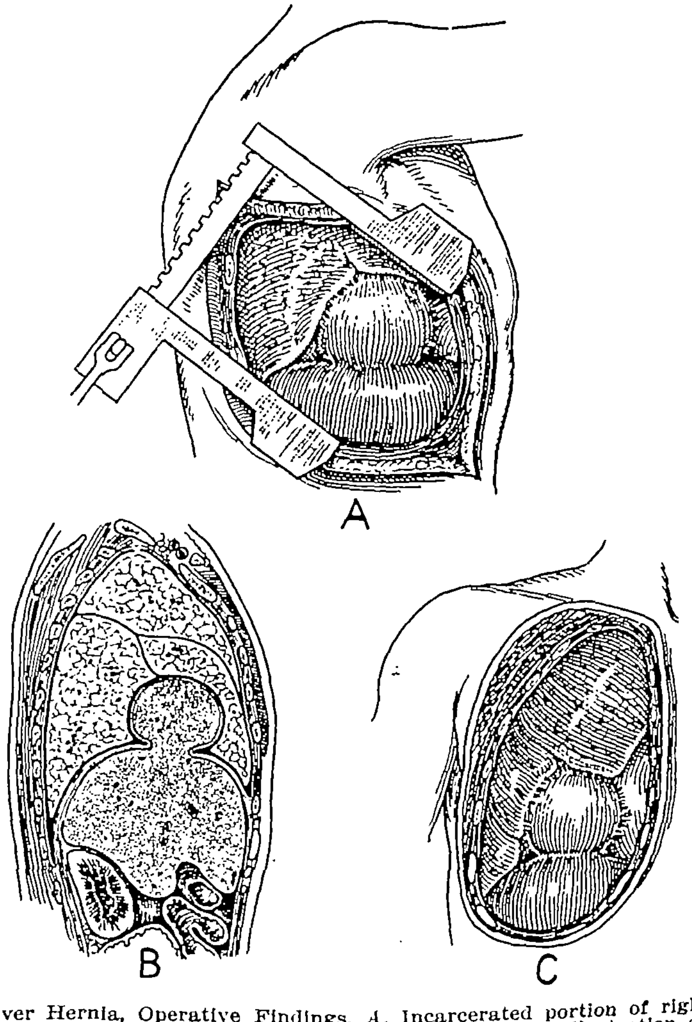

Fig. 2.—Liver Hernia, Operative Findings. *A*, Incarcerated portion of right lobe of liver protruding through central dome of diaphragm. *B*, Diagrammatic illustration of anatomic relations of liver, viewed in sagittal section through the hernia. *C*, Diagrammatic illustration of lesion viewed from in front.

liver protruding through the diaphragmatic tear. Keene and Copelman[8] reported delayed herniation of the liver and gall bladder in a severely injured paratrooper, but symptoms occurred soon after the trauma. The present case (as well as most of the others previously reported) was not diagnosed prior to operation. It is presented to stress the importance of considering diaphragmatic herniation of the liver in the differential diagnosis of rounded masses contiguous with the right diaphragm. Diagnostic pneumoperitoneum with roentgenographic study could be expected to aid in making the diagnosis.

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UNUSUAL DIAPHRAGMATIC HERNIA WITH DISPLACED LIVER

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TRAUMATIC diaphragmatic hernia with displacement of the liver is of uncommon occurrence. Harrington reported a series of 430 diaphragmatic hernias of all types and only two had livers displaced within the thorax. One of his cases was similar to the one reported herein, in that the right side was involved. Our case presented a double hernia with a diaphragmatic tear and an additional intercostal hernia. Organs involved were the liver, gall bladder, and hepatic flexure of the colon.

CASE REPORT

Mr. O. A., a 59-year-old white man, entered the Veterans' Hospital March 25, 1950, with a complaint of right chest pain which had been present since World War I. The pain had been dull and constant, and during the last several months had been seemingly progressive. Inspiration and walking aggravated it. It had been accompanied by a second pain over the precordium during the last three months which was described as being mild. He had never had any ankle edema, but was troubled with a frequent dry cough. Inasmuch as he had been diagnosed as having tuberculosis in 1918, he was concerned that this disease might be the offender. Inconspicuously, he told of a stab wound in the lower right anterior chest which he received eighteen years ago and had healed promptly.

The family history and past history were irrelevant.

Physical examination revealed an obese male of the stated age weighing 190 pounds. He was not orthopneic, and appeared comfortable. The findings were essentially limited to the chest. There was dullness over the lower half of the right thorax, and no breath sounds could be heard there. The heart was not enlarged to percussion, and the sounds were normal. There was a healed stab wound in the right seventh anterolateral interspace which had a ''mushy'' feel, and presented a defect in the intercostal muscle. There was no venous distention and no peripheral edema.

Hematology studies preoperatively showed a red cell count of 4.36 million, white cells 6,650, hemoglobin 12.2 Gm., and a normal differential smear. The sedimentation rate was 3 mm. per hour. Urinalysis was normal. Five sputum examinations were negative for tuberculosis organisms. Three stools were negative for ova and parasites. Skin tests for blastomycosis, coccidiomycosis, histoplasmosis, and echinococcus were negative. Bromsulphthalein test of the liver showed 10 per cent retention in 45 minutes.

A roentgenogram of the chest showed a slightly lobulated mass in the entire lower half of the right thorax continuous with the diaphragm. The heart was moderately displaced to the left. Barium studies of the intestinal tract revealed a loop of hepatic flexure of the colon above the expected level of the diaphragm and anterior to the mass. A diagnostic pneumoperitoneum revealed some of the air to have ascended into the right thorax partially surrounding the mass. The roentgenologist interpreted the mass to be liver, but could not rule out a mass within the organ itself. Roentgenograms of the gall bladder revealed nonvisualization.

Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion and policy of the Veterans Administration.

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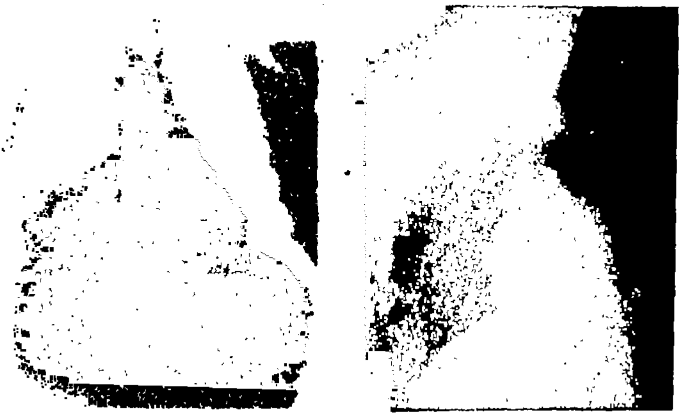

Fig. 1.—Preoperative roentgenograms. Posteroanterior view shows pneumoperitoneum.

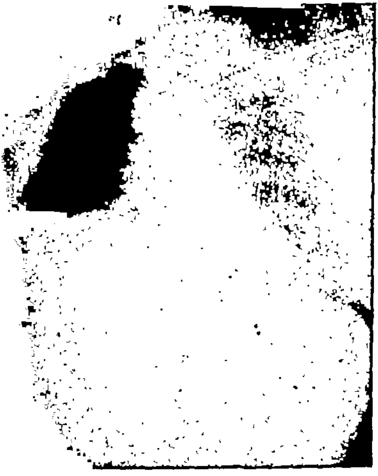

Fig. 2.—Postoperative roentgenogram with liver replaced and phrenic paralysis.

On April 25, 1950, the patient was explored through a right posterolateral incision over the bed of the seventh rib under endotracheal nitrous-ether-oxygen anesthesia. Three-fourths of the liver, including its entire right lobe, gall bladder, and hepatic flexure of the colon were found in the right thorax completely displacing all lung tissue from the lower half. Adhesions were dense but separated without difficulty. The liver and colon were returned to the abdomen, taking about five minutes to return the former organ by rotating and flexing it 90 degrees. No embarrassment to the heart was noted in this reduction. The right phrenic nerve was crushed, and the tear in the diaphragm sutured with interrupted silk. There was a 4-centimeter hernia in the seventh interspace where the colon had emerged at the stab wound scar. This was covered only by skin but was easily approximated by tacking the diaphragm against it. The lung expanded well and the pleural cavity was connected to an underwater trap by two right-angle drainage tubes. One thousand cubic centimeters of blood were given during the operation.

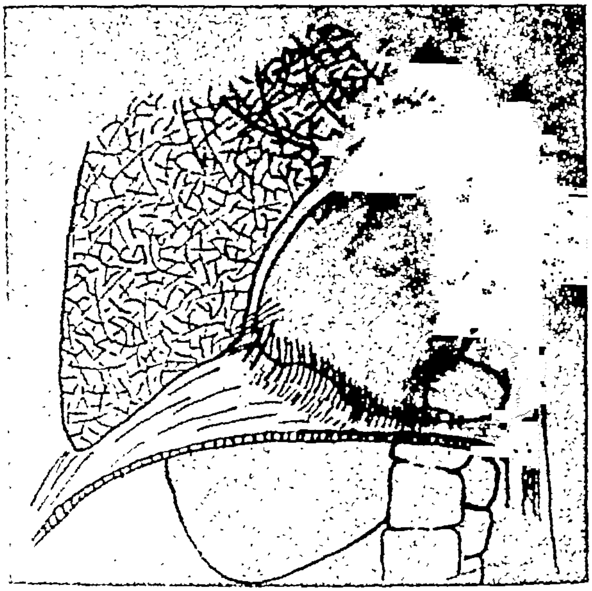

Fig. 3.—Operative findings—liver, gall bladder, and colon displacing lung through diaphragmatic and intercostal hernias.

Postoperative convalescence was uneventful. The patient was sitting in a chair on the second postoperative day and walking the third day. Within two weeks he remarked that he no longer was dyspneic and felt better than he had for years. No ventilation studies were done on this patient. He was discharged May 10, 1950.

DISCUSSION

The diagnosis of this case offered many problems. The presence of a seemingly lobulated mass in the lower thorax entails a differential diagnosis of tumors and cysts of the lung, liver, diaphragm, and mediastinum. The evidence of communication between the peritoneal and right pleural cavity was pure happenstance, and enhanced the effectiveness of the pneumoperitoneum in establishing the fact that this was a hernia. One normally expects to find a complete seal of the opening, particularly after many years have passed. Whether one was then dealing with a mass in the liver would depend on what the surgical exploration showed.

Symptoms in this case were limited to embarrassment of the cardiovascular system. The colon was not obstructed in its displacement. Although we did not have any preoperative diagnostic information on cardiac output, the patient demonstrated moderate dyspnea on exertion which was relieved by the operation. One can visualize the considerable impairment on the heart produced by the torsion and angulation of the inferior vena cava. The sedentary life of this patient probably helped him from evidencing any real cardiac failure.

Operative problems were overcome largely by (1) a thoracic approach, and (2) temporary relaxation of the diaphragm produced by crushing the phrenic nerve. Replacement of a large organ as the liver must be done gradually to obviate any severe taxation on the input to the heart. Suture of the large rent offers no problems if none of the atrophic muscle fibers of the diaphragm are sacrificed in the dissection. The intercostal hernia offered no technical problems and was more an interesting additional finding.

CONCLUSIONS

1. A traumatic hernia of the right diaphragm is reported which presented displacement of the major portion of the liver, gall bladder, and hepatic colon. It was accompanied by an intercostal hernia containing the loop of colon.

2. A liver displaced within the thorax can produce considerable cardiovascular impairment of function, and should be corrected early if possible.

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HOMOTRANSPLANTATION OF TRACHEAL SEGMENTS PRESERVED BY LYOPHILIZATION; AN EXPERIMENTAL STUDY

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THE use of tracheal homografts has been suggested as a possibility in repairing circumferential defects of the trachea. The application of such a procedure becomes apparent since resections of the trachea are feasible in the treatment of malignant disease primary in the trachea or secondarily affecting the trachea from adjacent structures.

Attempts to bridge defects in the trachea and primary bronchi by tubes of glass, stainless steel, or Vitallium has been attempted by Daniel.[1] He found that a viable tissue cover formed about the tube and in some animals, well-differentiated cartilaginous rings were formed in the developing tissue about the tube. Longmire[2] reported the use of a Lucite tube in repairing a tracheal defect in a patient with good results. Clagett and his co-workers[3] used Polythene tubes and most of the dogs did well, but in some instances the tubes came loose or collapsed. Successful autografts of segments of bronchus have been reported by Carter and Streider.[4] They also reported successful results with homografts, but in these cases stenosis of the bronchus was the great problem. Ferguson, Wild, and Wangensteen[5] have reported unsuccessful results at attempts to reconstruct short tracheal segments by means of autografts, homografts, aortic grafts, and tantalum gauze. However, these grafts were studied only for short periods of time.

Tracheal homografts can become feasible if methods are developed by which tracheal segments can be secured and stored in a relatively simple manner. The preservation of tissue by lyophilization was first suggested by Webster[6] in 1943. He attempted to transplant skin with poor results. In those tissues which can be transplanted successfully by host replacement, lyophilization is an ideal method of preservation because they can be kept at room temperatures for indefinite periods of time.[7]

The purpose of this experimentation was to determine the feasibility of lyophilizing tracheal segments to be used as satisfactory homografts in animals.

METHODS OF STUDY

Preservation of Tracheal Segments.—The tracheas to be preserved were taken from animals which had been sacrificed. They were placed in a lyophilizer, dried at a temperature of –15° to –25° C., and kept under vacuum (0.001 mm.) from seventy-two to ninety-six hours, removed, and placed in sealed jars. They were kept at room temperature until time of transplantation.

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Implantation of Tracheal Homografts.—Eight dogs were studied in this experiment. Under intravenous nembutal anesthesia, a linear incision was made in the midline of the neck, and the strap muscles were separated to expose the trachea. A portion of the trachea ranging from 2 cm. to 10 cm. in length was resected. A homograft of suitable size was hydrated with physiologic saline and sutured into place. Each animal was examined endoscopically at weekly intervals. Each animal was autopsied at appropriate intervals, and the site of transplantation examined.

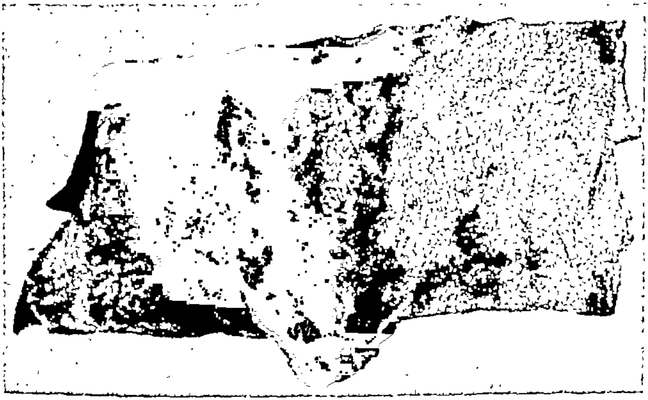

Fig. 1.—Lyophilized tracheal homograft before hydration.

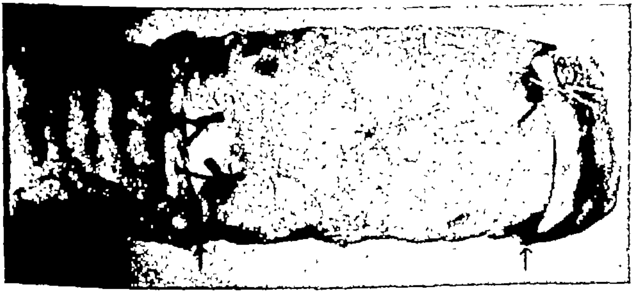

Fig. 2.—Dog No 4-10-50. Lyophilized homograft ten days after transplantation. Storage time thirty days.

RESULTS

Tracheal segments subjected to lyophilization are in all respects devitalized segments. They have the appearance of rigid tubes (Fig. 1). After hydration with physiological saline, they take on the appearance of normal tracheas.

Results of homotransplantation of lyophilized tracheas are summarized in Table I. There was no infection locally or in the mediastinum. Postoperatively all wounds of the trachea healed by primary intention. Endoscopically during the first two weeks postoperatively, marked congestion was noted in the area of the homograft. This increased vascularity decreased after the graft had healed and was not seen after three weeks. Healing occurred in all tracheal homografts except in one animal which developed pneumonia, and a severe necrosis was noted in the graft wall (Fig. 2). The grafts healed by replacement of the graft by fibrous connective tissue with loss of the cartilaginous rings (Fig. 3). In some instances, there was a tendency toward new cartilage formation within the graft. There was some torsion in the trachea noted, but no stenosis was noted in the grafted areas.

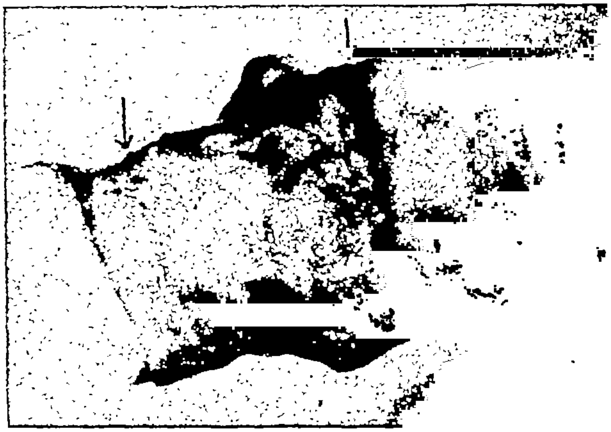

Fig. 3.—Dog No 4-20-50. Lyophilized homograft ninety days after transplantation. Storage time ninety days.

TABLE I. RESULTS OF TRANSPLANTATION OF LYOPHILIZED TRACHEAL HOMOGRAFTS IN DOGS

DOG NO.	LENGTH OF TIME GRAFT PRESERVED (DAYS)	LENGTH OF LIFE POSTOPERATIVELY (DAYS)	CAUSE OF DEATH	FATE OF GRAFT
4-1-50	10	8	Sacrificed	Excellent
4-5-50	20	15	Sacrificed	Excellent
4-10-50	30	10	Pneumonia	Necrosis of graft
4-15-50	60	30	Sacrificed	Excellent
4-20-50	90	90	Sacrificed	Excellent
4-25-50	120	90	Sacrificed	Excellent
4-30-50	15	90	Sacrificed	Excellent
5-1-50	8	120	Sacrificed	Excellent

DISCUSSION

In lyophilization, the tissue is dried in vacuum by reducing its water content as ice instead of evaporating it as a liquid. This process reduces it to its lighter and less bulky state, allowing it to be stored at room temperature without impairing its quality and value. The proteins are preserved in a dried state so that these transplants act in the same manner as fresh homografts or homografts stored in a refrigeration medium, The healing of lyophilized tracheal homografts occurs by replacement of the graft by fibrous connective tissue. A slight necrosis occurs within the wall of the graft during the first two weeks, but this does not interfere with the ultimate healing of the graft. Epithelium regenerates along the lumen of the graft by replacement, and there is some tendency toward cartilage formation in its wall so that a satisfactory bridging is obtained.

Primary suture of the trachea as shown by Maisel and Dingwall,[8] Scott,[9] Nach and Rothman,[10] and Jackson and his co-workers[11] is superior to homografting. When extensive segments of the trachea are to be resected, homografts may be necessary to bridge such defects. Homografting is feasible only when grafts are preserved and can be used as satisfactory grafts.

CONCLUSIONS

1. The use of preserved homografts by lyophilization has been described.
2. The healing of lyophilized tracheal homografts takes place by replacement of the graft by connective tissue
3. Homografts may be necessary to bridge defects where extensive segments of trachea are resected.

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BRONCHIOGENIC NEOPLASM MASQUERADING AS ALVEOLAR CELL CARCINOMA

With Report of a Case Clarified Only by Autopsy

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SIGNIFICANCE of human alveolar cell tumor has been debated for years. According to Ikeda[26] and Neubuerger and Geever,[36] this condition was probably first described by Malassez in 1876 as encephaloid carcinoma. Since then a considerably widening nomenclature has developed; and now includes pulmonary adenomatosis, suggested by Drymalski, Thompson, and Sweany,[15] by Sims,[47] by Simon,[44, 46] by Richardson,[41] by Hildebrand,[25] and by Bubis and Erwin;[9] carcinosis, advocated by Bonne;[6] pulmonary mucous epithelial hyperplasia, favored by Taft and Nickerson;[49] diffuse primary alveolar carcinoma of the lung, advanced by Simon;[45] alveolar cell tumor, nominated by Neubuerger and Geever;[36] primary multiple alveolar cell tumor, proposed by Neubuerger;[35] and pulmonary alveolar adenomatosis, preferred by Wood and Pierson.[54] Both Ikeda[26] and, more recently, Delarue and Graham[14] have suggested the term alveolar cell carcinoma as most appropriate.

The number of published cases, regardless of title, exceeded seventy when recently tabulated by Simon.[45] But antemortem recognition was claimed for only three in this entire group, and solely by "microscopic examination of surgically obtained tissues," according to Weir.[51] However, the latter has also described an additional patient in whom aspiration biopsy was diagnostic, and considers this the first correct preoperative diagnosis ever reported for this condition.

After reviewing the literature, we shall discuss a further interesting case in which preoperative exfoliative studies, preoperative bronchoscopic biopsy, microscopic surgical specimen examination, and gross autopsy findings were all compatible with primary alveolar carcinoma. Unexpectedly, however, microscopic postmortem study revealed bronchiogenic malignancy, with pattern of metastasis entirely indistinguishable from that commonly described in alveolar cell tumor.

We consider such a report instructive for two reasons. In the first place, apparently all diagnostic criteria had been fulfilled before any surgical procedure was carried out. An even more important, our eventual findings appear to cast some doubt upon the possibility of pure alveolar carcinoma actually existing in primary form.

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CONTROVERSIAL PERSISTENCE OF HUMAN ALVEOLAR EPITHELIUM

Although alveolar epithelium is usually considered prerequisite to development of primary tumor in this location, embryologists and pathologists do not completely agree concerning its persistence after the fifth fetal month.

Bell[5] believes that partial disappearance at that time assists in future oxygen-blood interchange, but *feels that certain cells survive and may subsequently* form a continuous layer under proper stimulation. He has seen evidence of the latter in chronic passive pulmonary congestion, and lipid pneumonias, and states that hyperplasia may "arise by multiplication of pre-existent alveolar epithelial cells, and not as an ingrowth of bronchial epithelium." Wood and Pierson,[54] Ikeda,[26] Casilli and White,[10] and Miller[32] also agree; the latter states that "evidence points to a continuous epithelium lining the pulmonary alveoli."

In further confirmation, it is recalled that Slye, Holmes, and Wells[48] reported spontaneous development of alveolar tumors among mice in 1914. Murphy and Sturm[34] produced similar lesions after application of coal tar distillates to the skin. Subsequently Grady and Stewart[20] reduplicated these findings among inbred strains by subcutaneous injections of 1, 2, 5, 6—dibenzanthracene; it seems unlikely that any of these animal tumors would have developed unless alveolar epithelium was originally present.

Fried[16] considers that human bronchopulmonary carcinoma arises from alveoli, bronchial mucosa, or bronchial glands; and, therefore, accepts the fact that alveolar epithelial cells persist after birth. Cooper[11] and Karsner[30] agree with the latter conclusion, although Karsner concedes that exact cellular structure is not clear. Neubuerger and Geever[18] have demonstrated persisting alveolar epithelium in certain chronic pulmonary conditions such as silicosis and tuberculosis, and following irritation by chemicals, oils, gases, and toxins. They also refer to Brachet's concept that *all* germ layers may develop unusual reactions after stimulation; the latter possibility, if confirmed, would permit development of neoplasm from pulmonary mesenchyme alone.

Among those opposing survival of alveolar epithelium, Herbut[22] is perhaps the most emphatic. Basing his conclusions upon study of selected cases from 6,500 consecutive autopsies, he asserts that apparent alveolar hyperplasias are actually due *to downgrowth of encroaching bronchial mucosa, and can always* be shown to merge with the latter by careful study; both he and Frissel and Knox[17] feel that true bronchiogenic carcinoma may reproduce the exact peripheral pattern often described as primary alveolar tumor. Arkin and Wagner[3] consider all pulmonary carcinoma bronchiogenic, and feel that malignancy cannot derive from alveoli, since the latter retain no persistent epithelial lining. Neubuerger,[35] Barnard and Day,[4] *and* Geever, Neubuerger, and Davis[18] are satisfied that alveolar epithelium disappears before birth, that it does not reappear in adult life, that replacement is made by mesenchymal septal cells and capillaries, and that carcinoma cannot develop from mesenchyme. Ham and Baldwin[21] classify alveoli as "merely spaces in tissue derived from cellular mesenchyme" and *therefore* incapable of *originating* tumor. *Moore,*[33] Weller,[52] and Jordan and Kindred[29] find little evidence of residual adult epithelium, while

D'Aunoy, Pearson, and Halpert[13] give such negligible consideration to the latter's survival that the alveolus is not even mentioned as a possible site of lung tumor.

Other investigators have been in doubt. Macklin[31] summarized a round table discussion among various authorities in the middle '30s, from which no definite conclusions were forthcoming, one group feeling that a complete lining persisted, although possibly invisible to usual means of examination in normal tissue, another group accepting evidence of occasional residual surviving cells, while a third group was convinced of complete disappearance and permanent replacement by mesenchyme.[31] Taft and Nickerson[40] concede the possibility of adult epithelium, but do not entirely exclude a bronchial source; Boyd[7, 8] believes all reported alveolar hyperplasias are probably bronchial in origin, while Jaffe,[28] Anderson,[2] and Herbut[23] are undecided.

One is therefore faced with lack of complete agreement concerning postnatal alveolar epithelium. Unless he accepts proof of the latter, he cannot reasonably hold that primary alveolar tumor may develop therefrom. However, since a persisting membrane has definitely been reported by several authorities,[32 et al.] it does appear to us that included cells are capable of providing the necessary framework for localized hyperplasias, and perhaps even malignancy.

ETIOLOGY OF ALVEOLAR METAPLASIAS

In the preceding section, various investigators have been indirectly quoted in reference to carcinogenic properties of certain irritants, and to probable stimulating effects of chronic pulmonary diseases, in production of alveolar hyperplasias. Richardson,[41] while doubting frequent involvement of this type, agrees that occasional lesions may range from incidental autopsy findings to pathology sufficient to produce death.

In further confirmation, Seelig and Benignus[43] have found that inhaled chimney soot could produce generalized pulmonary carcinoma in cancer-sensitive mice; while, among a genetically resistant strain, pure alveolar involvement was suggested. In 1920, Winternitz, Smith, and McNamara[53] stimulated alveolar proliferation in rabbits by insufflation of 0.25 per cent HCl, as a result, alveoli developed an entirely new epithelial layer, similar to that seen after influenzal pneumonias, but involving bronchi as well. Young[55] has demonstrated related alveolar changes after intrapleural irritation by paraffin, bile, and other salts. Ikeda[27] created circumscribed nodular pulmonary lesions in rabbits through tracheal administration of various liquids; by use of cod liver oil, for example, alveolar epithelization was demonstrated within two weeks. Furthermore, Norris[37] discovered comparable changes in guinea pigs after inoculation of fluid aspirated from a patient with pneumococcus pneumonia, and quotes identical results achieved by Grumbach through use of diphtheroid bacilli recovered from a patient with Hodgkins disease. Related, although unexplained, findings have also been reported in mice and horses, according to Bubis and Erwin.[9]

Although Frissell and Knox[17] do not accept proof of alveolar carcinoma, they have conceded that metaplasia in immediately adjacent bronchial mucosa may result from various irritations; the latter seem to include influenza, war gas, tuberculosis, products of tar and cigarette combustion, radioactive mine dusts, and others. Heuper[24] does not restrict possibility of such changes to the bronchus, and in 1950 concluded that identical pathology might also occur within the lung field itself.

These various investigations, therefore, suggest that significant changes in alveolar membranes may well occur after specific irritation. And perhaps most likely, as intimated by Delarue and Graham[14] in referring to Andervont's observations in mice, in those with genetic tendency to such. This factor of predisposition and susceptibility is also emphasized by Ikeda[26] and Alexander and Chu,[1] who relate it to a chain of progressive abnormalities consisting of (a) hyperplasia, (b) benign adenomatosis, and (c) alveolar carcinoma. No evidence yet incriminates viral etiology in human malignancy of the latter type, although a virus is apparently responsible for Jaagziekte, an ovine illness in many ways similar to alveolar cell tumor in man.

Jaagziekte has variously been described as epizootic adenomatosis, pulmonary adenomatosis of sheep, verminous pneumonia, and progressive pneumonia of sheep. First reported by Eber in 1891,[47] and originally considered by M'Fadyean to be caused by parasitic lung worms, it is now endemic in South Africa, Iceland, England and the Rocky Mountain regions in and about Montana. Cowdry,[12] in 1925, eliminated spirochetes, nematodes and coccidiosis as responsible agents; and today a virus etiology is generally accepted. Incidentally, no authenticated instance of transmission from sheep to shepherds has yet been described.[44]

Characteristic histopathological structure in Jaagziekte closely simulates that seen in human beings reported dying of alveolar cell carcinoma. It includes multicentric pulmonary foci; tumorlike reproduction of alveolar structure with columnar, occasionally papillary, and nonciliated cells; relative absence of mitosis; and general infrequence of stromal invasion. The lung pattern, according to Geschickter and Dennison,[19] duplicates that seen in mice after cutaneous coal tar applications. Metastasis, discovered in about 50 per cent of cases labelled human alveolar carcinoma, has been reported only once in Jaagziekte,[54] however.

Although difficult to transmit artificially, Jaagziekte appears to spread with great rapidity when healthy sheep are confined with infected flocks. Dungal, according to Wood and Pierson,[54] suspected that individual susceptibility varied according to genetic strain of the herd involved; this observation might further explain spotty and unpredictable development of somewhat similar alveolar tumors in man. It is not surprising, therefore, that numerous investigators have postulated a relationship between Jaagziekte and human alveolar carcinomatosis, although a virus does not appear to be the responsible agent in the latter condition.

PATHOLOGY OF CASES DESCRIBED AS ALVEOLAR CELL CARCINOMA

Those[26, 28, 45, 13, 54, 44, 14, 51 et al.] who have reported autopsied cases agree generally upon gross and microscopic findings. Both a diffuse form, first mentioned by Musser in 1903,[38] and a multiple nodular type are described, each usually occurring in the same individual, and often in the same lung.

overlying an apparently consolidated or atelectatic right lung; contralaterally, a diffuse soft infiltration was seen. Although two sputum examinations failed to reveal tubercle bacilli, provisional diagnosis of pulmonary tuberculosis and pleurisy with effusion was made. Hospitalization was accepted Dec. 19, 1949.

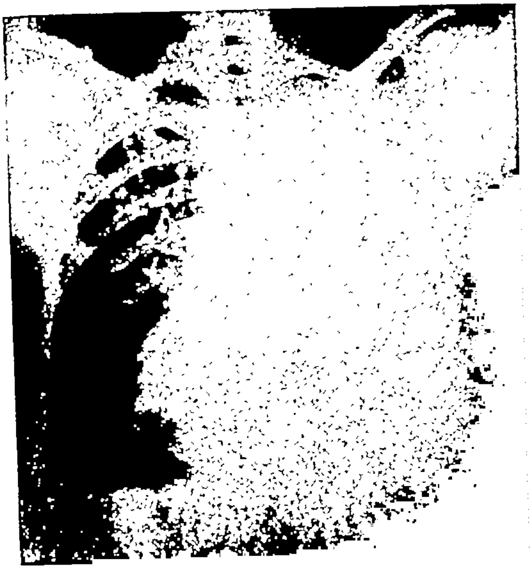
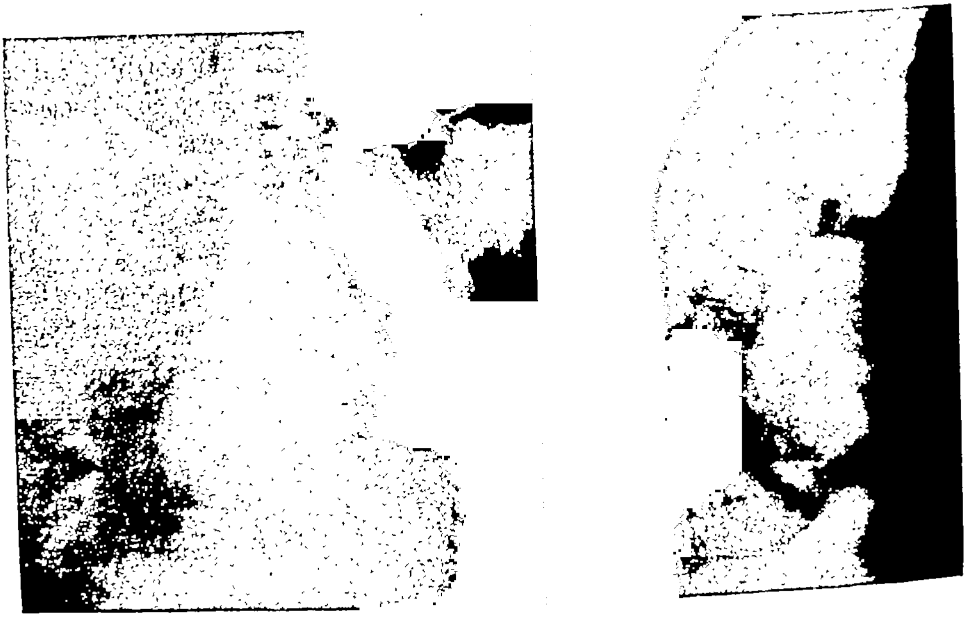


Fig. 1.—Hospitalization chest film, Dec. 20, 1949.



A.

B.

Fig. 2.—Posteroanterior and lateral chest films after thoracentesis. *A*, Feb. 1, 1950; *B*, Jan. 23, 1950.

The patient was moderately febrile on admission, but without significant respiratory complaint. Painless facial twitching was apparent; right Babinski and bilateral Oppenheims were present. Sensation was normal. No lymphadenitis was discovered, although liver edge and splenic tip were palpable. The thorax was asymmetrical, with right-sided lag. Pulmonary physical findings were consistent with right pleural effusion or empyema, while râles were audible throughout the left chest only. The heart was of normal size, shape, and position; without irregularity, but with simple sinus tachycardia. A soft, nontransmitted mitral systolic murmur was not associated with palpable thrill. Blood pressure was 120/80. Vital capacity 1,300 c.c. or 33 per cent of anticipated normal.

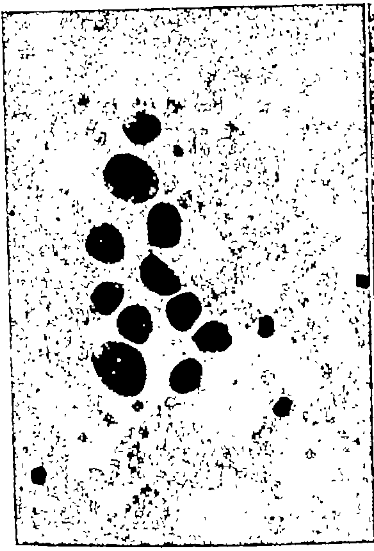


Fig. 3.

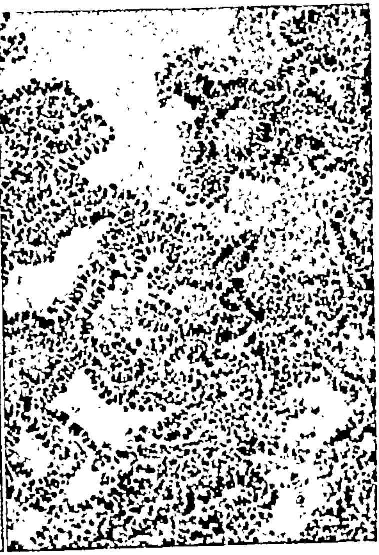


Fig. 4.

Fig. 3.—Exfoliative study of bronchoscopic aspirations.
Fig. 4.—Biopsy from lower lobe bronchus, Jan. 11, 1950.

Admission roentgenogram suggested right-sided tuberculous or nontuberculous pathology (possibly malignancy complicated by right pleural effusion or empyema) and a progressing contralateral lesion (Fig. 1). Old Tuberculin was positive (1:10000 dilution); histoplasmin and coccidioidin skin tests were negative. Concentrated sputum examination failed to reveal tubercle bacilli. A mild macrocytic (MCV 111), normochromic (MCH 32) anemia was present. White blood count totalled 10,400, with 75 polymorphonuclears (of which 73 were segmented forms), 12 lymphocytes, 10 monocytes, 1 eosinophile, and 2 basophiles. Sedimentation rate was not accelerated; urinalysis showed no abnormality; serology was negative.

Certain further diagnostic procedures then carried out may be summarized as follows:

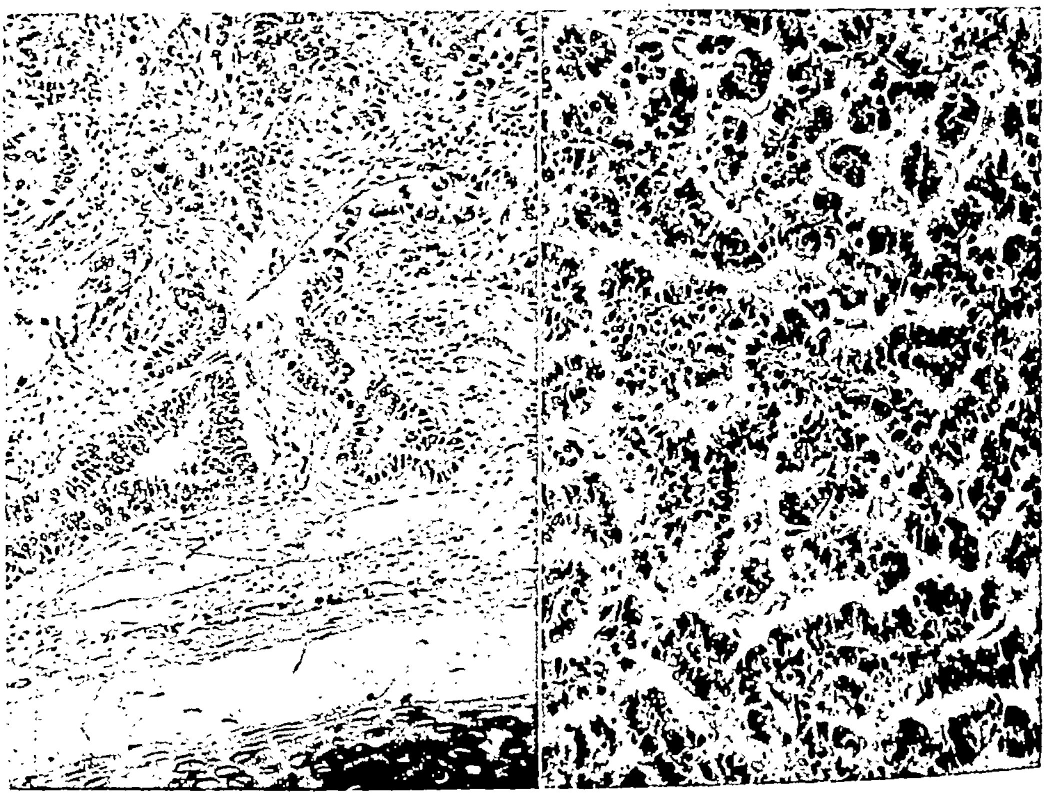
(a) *Multiple thoracenteses*: Thin wine-colored fluid was aspirated on numerous occasions. Tubercle bacilli were absent by concentration in eight specimens, by culture in eight, and by animal inoculation in three. No other organisms were cultured in six examinations. Eight fluid specimens failed to show neoplastic cells. Specific gravity varied between 1.022 and 1.028, protein between 150 mg. and 5 Gm. Cells ranged from 1,350 to 3,400 per cubic millimeter; of these, polymorphonuclear forms fluctuated between 58 and 72 per cent, with the remainder largely lymphocytes. Sugar was variously reported between 8 and 35 mg. per cent.

Liver: The surface, particularly of the upper portion, was covered with neoplastic nodular tissue. There were some subcapsular areas of white neoplastic tissue. The cut surface showed a rather small number of small-sized scattered round white neoplastic nodules. These averaged about 3 mm. in diameter. Most neoplastic involvement of the liver was capsular. The gall bladder and bile ducts were normal.

Pancreas: Normal.

Gastrointestinal Tract: No abnormality was noted in the stomach or intestine.

Retroperitoneal Tissues: The neoplastic extension through the diaphragm, and to the undersurface of the diaphragm, extended down on the anterior surface of the peritoneum lining the posterior abdominal cavity in the area anterior to the right kidney. As has been mentioned, there was involvement of both adrenals. There likewise was involvement of the lymph nodes of the retroperitoneal area along the aorta. There seemed to be fairly definite involvement of the left sympathetic trunk. Involved lymph nodes lay anterior to the right sympathetic trunk.



A. B.

Fig. 9 A and B.—Microscopic section appearance of bronchial mucosa and parenchyma of right lung.

Thyroid: The thyroid gland was normal.

Anatomical Diagnosis.—

1. Diffuse lobar consolidation of entire right lung and upper lobe of left lung, with confluent lobular consolidation of lower lobe of left lung, by an alveolar carcinoma of pulmonary origin.
2. Neoplastic extension through diaphragm, with peritoneal and omental involvement.
3. Neoplastic involvement of adrenals and retroperitoneal lymph nodes.
4. Metastatic neoplastic lesions of liver.
5. Old organizing right hemothorax.
6. Recent hemoperitoneum.

MICROSCOPIC EXAMINATION

Lung: Several sections from the lower lobe of the right lung disclosed extensive alteration of bronchi by neoplastic tissue. The lumens were narrowed. The normal mucosa was *replaced by a mass of neoplastic tissue. Neoplastic tissue streamed through the wall and* diffusely into surrounding lung tissue. Microscopically these findings were rather typical of those ordinarily seen in bronchiogenic carcinoma (Fig. 9*A*). In the area of the replaced bronchial mucosa the tumor tended to be of an adenocarcinomatous structure. As it spread into the surrounding tissue it became of an alveolar type, into which there were frequent papillary projections. Groups of such alveoli were separated by fairly regularly distributed interlobular septa (Fig. 9*B*). Sections from the left lung disclosed lobularly distributed masses of similar appearing tissue, separated by some uninvolved lung tissue which was the site of extensive congestion and edema (Fig. 10). Sections from the left lung did not show bronchial involvement. The distribution of these tumor cells afforded ample justification for employment of the term "alveolar carcinoma." *However, their apparent origin from* bronchial mucosa in an area with the usual pattern of an adenocarcinoma suggested that the alveolar arrangement was a type or variant of adenocarcinoma of bronchiogenic origin, rather than a primary tumor of alveolar epithelium.

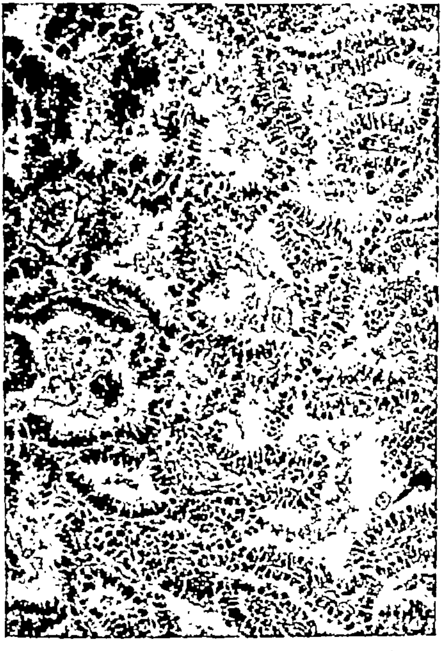


Fig. 10.—Microscopic section appearance of left lung.

Mediastinal Cyst: The cyst found between the esophagus and the hilus of the right lung was lined with ciliated columnar epithelial cells of the type ordinarily found in trachea and bronchi. *These formed spaces extending into smooth muscle bundles apparently represent-*

ing the wall of the esophagus. Extending tumor tissue from the neoplasm of the right lung was found in close proximity, but this appeared to be coincidental.

Heart: Coronary vessels showed minimum sclerosis. The epicardium was normal, myocardial fibers were of normal size and staining reaction.

Spleen: Normal.

Adrenal: There was extensive metastatic neoplastic tissue quite similar to that seen in the lung and tending to maintain the same alveolar distribution of cells. There was extension and infiltration into the surrounding fat and fibrous tissue where the pattern was more akin to that of the usual adenocarcinoma (Fig. 11).

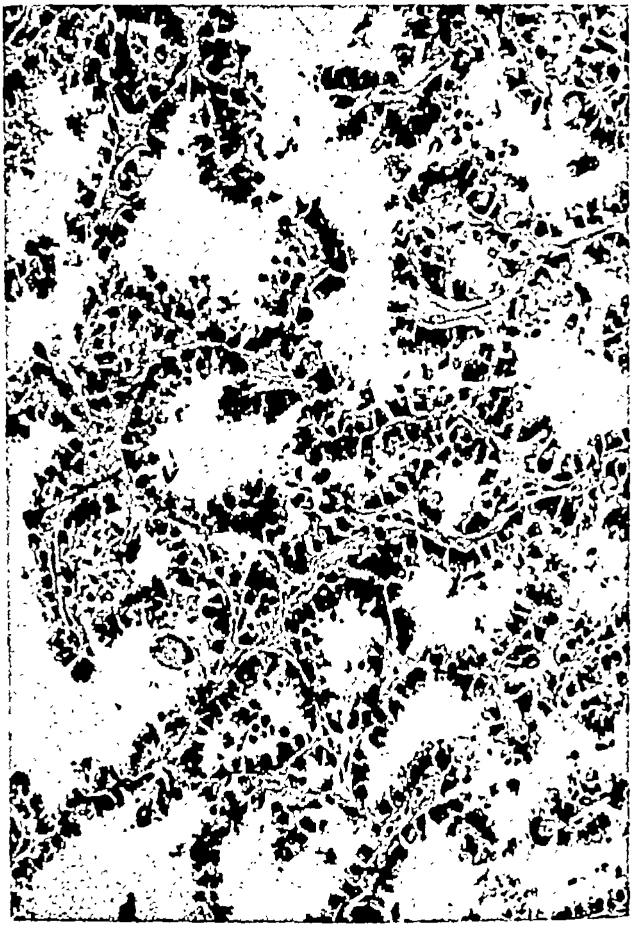


Fig. 11.—Appearance of adrenal metastasis.

Kidney: There was no evidence of neoplastic involvement. The general architecture, blood vessels, and glomeruli were normal. Tubules were normal or showed slight epithelial degeneration.

Prostate: Normal.

Liver: Hepatic cells were extensively shrunken with resultant widening of sinusoids. The liver contained an area of tumor tissue similar to that seen elsewhere, including some portions with alveolar grouping.

Pancreas: Normal.

Thyroid: Normal.

Sympathetic Ganglia: There was extensive neoplastic infiltration.

Microscopic Diagnosis.—

1. Bronchiogenic adenocarcinoma giving rise to diffuse alveolar carcinoma of lung, with metastases to adrenals, liver and retroperitoneal structures.
2. Bronchiogenic mediastinal cyst.

FINAL DIAGNOSIS

1. Bronchiogenic adenocarcinoma of lower lobe of right lung with diffuse alveolar carcinoma of right and left lung, and with extension through diaphragm involving peritoneum and omentum, adrenals, sympathetic ganglia and liver.
2. Old organizing right hemothorax.
3. Recent hemoperitoneum.
4. Bronchiogenic mediastinal cyst.

DISCUSSION

In spite of difference in opinion concerning persistence of human alveolar epithelium after the fifth fetal month, there is consensus that certain cells persist in relic form and, after irritation and stimulation, may readily be demonstrated. Especially following pneumonias, during passive pulmonary congestion, and in other conditions where edema separates the alveolar membrane from surrounding structures and aids in its identification.

Debate has naturally arisen over the possibility of discovering primary tumor in this area, with most investigators apparently feeling that carcinogens and irritants may occasionally produce a localized neoplastic change. Such is known to occur in laboratory animals, and similar irritation is also generally accepted as partially responsible for human carcinomas of the bronchiogenic type. Furthermore, sheep may develop a somewhat parallel alveolar lesion known as Jaagziekte, presumably caused by a virus, but apparently most frequently seen in flocks with genetic predisposition to such.

Although more than seventy cases of human alveolar cell carcinoma have been described under varying nomenclature, Herbut and others do not believe the tumor is primarily alveolar in origin and feel they have demonstrated direct communication between a responsible bronchial site and involved alveoli. Those favoring primary alevolar tumor as an actual clinical entity, however, point out certain characteristics which are said to clearly distinguish it from all other malignancies. These include (a) reduplication of alveolar pattern by cells whose morphology differs entirely from that of bronchiogenic carcinoma, (b) failure to identify primary neoplasm elsewhere, and (c) multicentricity.

With these criteria as a guide, our patient was considered to have primary alveolar cell carcinoma throughout most of his hospital residence. This impression was supported by sputum and bronchoscopic exfoliative studies, bronchoscopic biopsy, postresection specimen examination, and gross autopsy findings. He showed the reported lack of agreement between respiratory symptoms and extreme pulmonary involvement by roentgenogram; tuberculosis and extrapulmonary malignancy were apparently excludable; multicentric lesions, both of the diffuse nodular and massive consolidative types, were suggested by a variety of examinations. In short, until postmortem section study was completed, this case very closely simulated others reported as authenticated primary alveolar carcinomas; and, also important, was one in which diagnosis had apparently been made by us before any operative procedure was offered.

Lower extremity neuritis was a clinical finding not clearly understood during this patient's life but, in view of autopsy studies, can now be ascribed to retroperitoneal metastasis and involvement of major nerve trunks. Identification of nonpathogenic tubercle bacilli in a single preterminal sputum specimen was considered a "red herring," since we have seen four hospital cases in the past three years with an identical finding, but without evidence of clinically significant tuberculosis by autopsy.

Microscopic autopsy sections were crucial in this case. They disclosed a primary bronchiogenic adenocarcinoma which streamed into the lung field, with widespread tumor metastasis assuming an extremely striking and characteristic alveolar pattern. Our error in diagnosis underscores the fact that considerable caution should be employed in labelling any condition primary alveolar tumor, unless very careful search has excluded a primary bronchial site; even if the latter is not discovered, it may simply have disappeared under advancing metastatic invasion.

We do not feel that primary alveolar carcinoma is necessarily a nonexistent condition, but do find reason to support a contention that it is not as common as has been reported. Analysis of our case would appear to add further weight to an impression that alveolar tumors are frequently metastatic in type, and need not represent true primary malignancy.

SUMMARY

1. After reviewing various opinions concerning persistence of alveolar epithelium in the human being, it is considered that existence of a residual cell membrane of this type has been established.

2. Survey of the literature also indicates, although opinion is by no means unanimous, that metaplastic and even neoplastic changes may develop in alveoli after exposure to various irritants or carcinogens. This may well occur in those with genetic predisposition to such.

3. We have reviewed similarities in pathology and dissimilarities in etiology between this reported condition in human beings and Jaagziekte in sheep.

4. Characteristics said to identify human primary alveolar cell carcinoma include multicentricity of pulmonary lesions, reduplication of alveolar pattern by cells whose morphology is dissimilar to that seen in other malignancies, and failure to identify a primary site elsewhere, including bronchial mucosa.

5. A case is described in detail. Diagnosis of primary alveolar carcinoma seemed well established through histopathologic examination of bronchoscopic biopsy material, resected pulmonary tissue, and gross autopsy findings, as well as through exfoliative studies of bronchoscopic aspirations and sputum.

6. It is of interest that we believed a diagnosis of alveolar carcinoma had been made before surgical intervention; if this had finally been confirmed, C. K. would have represented the second such patient described in the literature to date.

7. Microscopic autopsy section study, however, definitely disclosed true primary bronchiogenic adenocarcinoma. Clearly identified metastasis was extensive to lung and other viscera, and had definitely assumed that pattern described as typical of alveolar cell tumor.

8. Study of this case, in reference to others previously reported, would infer that several of the latter might not have represented true primary alveolar carcinoma. Some support is offered Herbut's contention that none are of the latter type, and that true bronchiogenic origin was not established simply because of disappearance under metastatic invasion.

9. Naturally, we suggest no final conclusion, but do believe that report of this case may contribute to further clarification of that question.

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THE LATERAL COSTAL BRANCH OF THE INTERNAL MAMMARY ARTERY

BENJAMIN N. KROPP, PH.D.
KINGSTON, ONTARIO

IN DISSECTING room subjects there is sometimes encountered a thoracic artery arising from the internal mammary near its origin and running laterally downward on the inner wall of the chest. The apparent frequency of its occurrence, its occasional large size, and the absence of its mention in many anatomic books of reference are striking, especially in view of the obvious importance of such a structure in paracentesis and other surgical procedures of the thorax.

The artery was known to the older anatomists. Henle (1876) describes an arteria mammaria interna lateralis which arises from the internal mammary near its entrance into the thorax and descends on the inner surface of four to six upper ribs and anastomoses with the corresponding intercostal arteries. He quotes Heister (1730) as having seen it in a 3-year-old child as well as in adult subjects. Henle pictures it with a good two-color illustration, and states that in thoracentesis or by traumatic damage the artery may be injured, and dangerous hemorrhage result. Otto (1830) calls it ramus costalis lateralis, a name retained by Rauber-Kopsch (1941) and others. Macalister (1889) reports having seen it of enormous size in a case of obliteration of the dorsal aorta from pressure of an intrathoracic tumor. In his extensive study of the arterial system of the Japanese, Adachi (1928) gives data on its frequency of occurrence, and notes also that it may arise as a branch of the subclavian or supreme intercostal arteries. Although it has not been investigated from the comparative anatomic viewpoint, it is interesting that Morestin (1894) reports it widely present in certain monkeys. Bean (1905) found the artery present five times in twenty-eight American mullatoes. However, when seen and remarked upon by anatomists, it has usually been considered an anomaly of rarer occurrence than the data would indicate. The artery is known under a variety of names, summarized in Table I.

The oldest term applied to the artery seems to be lateral internal mammary artery, given by Heister in 1730 and adopted by Henle. That name, however, seems to imply a constancy of occurrence which the artery does not possess. *Lateral costal branch* of the internal mammary artery is adequately descriptive and is used in this report.

In the present study 112 white subjects, all but eight of them males, were examined. The artery was seen in but one female. No precise data are avail-

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From the Department of Anatomy, Queen's University Medical School.
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TABLE I.

NAME	AUTHOR
Lateral costal branch	Otto, 1830 v. Langer & Toldt, 1902 Cunningham, 1937 Rauber-Kopsch, 1941 Morris, 1942
Internal thoracic artery	Hodges, 1867
Lateral internal mammary artery	Henle, 1876 Morestin, 1894 Adachi, 1928
Retrocostal artery	Hellema, 1862 (cited from Henle)
Subcostal artery	Tiedemann (cited from Henle)
Lateral infracostal artery	Macalister, 1889 Toldt, 1926
Mammaire interne accessoire	Poirier et Charpy, 1902
Median intercostal artery	Hyrtl (cited from Poirier & Charpy)
Lateral thoracic artery	Bean, 1905

able as to age of this series, although almost all were over 60 years. In thirty-one individuals (27.6 per cent) the artery was present on one or both sides. When present on one side only it occurred more frequently on the left than on the right. The frequency of occurrence is summarized in Table II.

TABLE II.

Number of Subjects—112		
Artery present on right side only	10	8.9%
Artery present on left side only	15	13.4%
Artery present on both sides	6	5.3%
Total subjects in which artery present	31	27.6%

Adachi's data from 172 lateral chest walls are not significantly different from the above. When present bilaterally the lateral costal branch is not necessarily the same length on the two sides. The variation in length, as indicated by the frequency with which a given number of intercostal spaces were traversed, is shown in Table III.

TABLE III.

INTERCOSTAL SPACES TRAVERSED	1	2	3	4	5	6	TOTAL
Right side	2	3	4	5	1	1	16
Left side	3	6	4	5	1	2	21

When present, the lateral costal branch arises from the internal mammary artery at or immediately above the superior border of the first rib (Fig. 1). It passes downwards, backwards, and laterally from its origin, covered only by parietal pleura. In its course the artery approximates the midaxillary line, although it has been observed lying well forward toward the mammary line. Contrary to Adachi's observation, none of our specimens took origin from the subclavian or supreme intercostal arteries. Variations were seen, however, in its origin from the internal mammary; in one case it arose 1 cm.

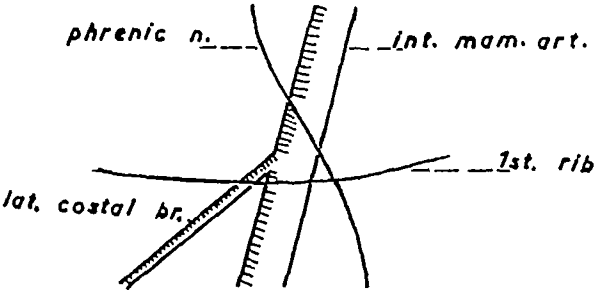

Fig. 1.—Right side. Diagram showing origin and direction of lateral costal branch of internal mammary artery, and usual relation to phrenic nerve and upper border of first rib.

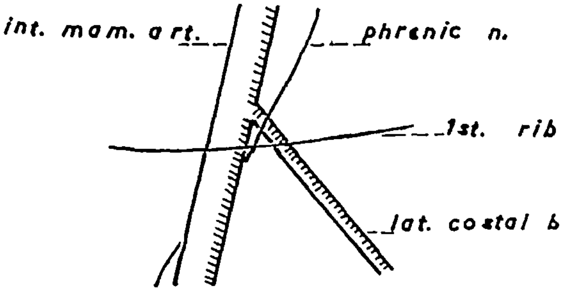

Fig. 2.—Left side. Diagram of occasional course of phrenic nerve over lateral costal branch.

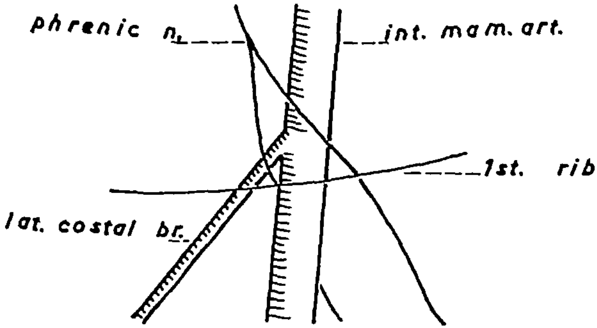

Fig. 3.—Right side. Diagram of usual course of branched phrenic nerve with relation to lateral costal branch and internal mammary artery.

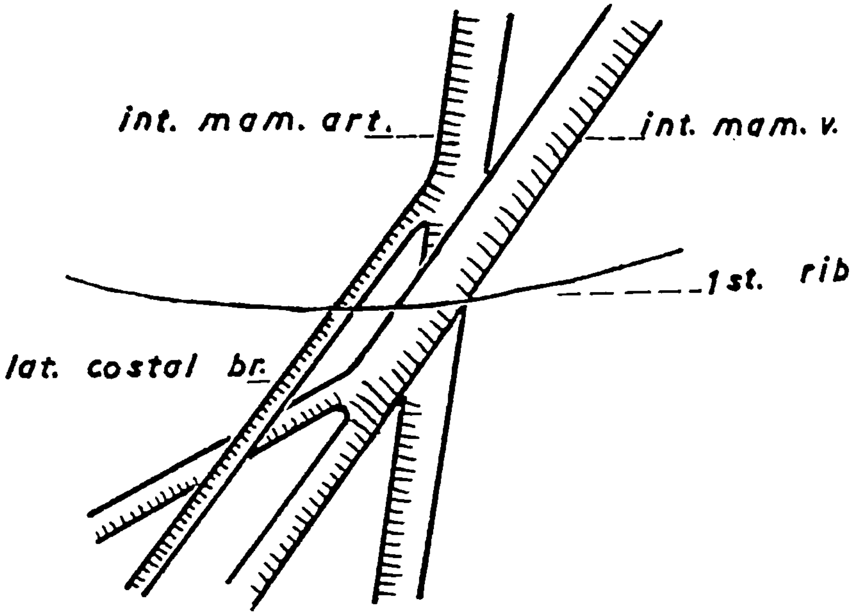

Fig. 4.—Right side. Diagram showing relation of internal mammary vein to lateral costal branch.

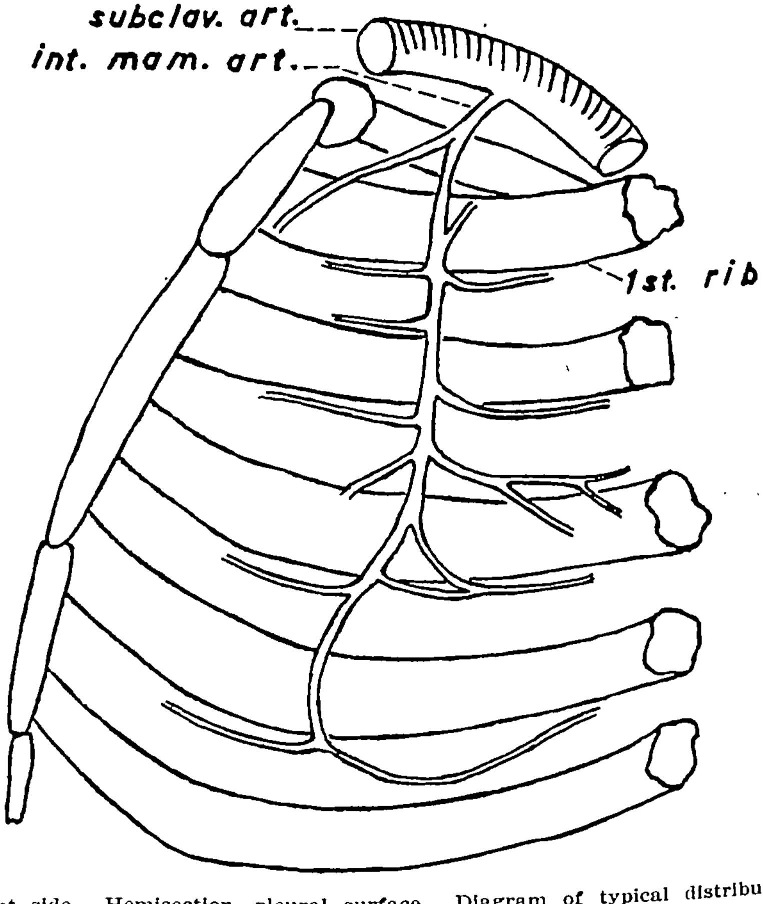

Fig. 5.—Right side. Hemisection, pleural surface. Diagram of typical distribution pattern of lateral costal branch.

above the upper border of the first rib and in another at the level of the lower border. On the right side the usual relation to the phrenic nerve is seen (Fig. 1), the nerve passing over the internal mammary and above the lateral costal branch. In two instances on the left side and once on the right, the nerve passed over the lateral costal branch and deep to the internal mammary artery behind the first rib (Fig. 2). Fig. 3 represents a case in which the divided nerve sent one small branch under the internal mammary after passing over the origin of the lateral costal, while the larger nerve maintained its usual relation to the internal mammary artery. No case was observed in which the phrenic nerve passed under both arteries or under the lateral costal and over the internal mammary.

The caliber of the artery varies with its length and the number of its anastomosing branches. When it extends over six or more intercostal spaces, it has been observed as large as the internal mammary. When viewed from within the thorax the lateral costal branch may be traced with ease beneath the pleura (Fig. 5), with branches given off in each intercostal space both anteriorly and posteriorly, usually at the lower border of the ribs. Branches may pass downwards over one or two ribs, however, before becoming continuous with the intercostal arteries. Occasional small twigs merely cross the pleural surface of a rib, or penetrate the rib as nutrient arteries. Near its termination the lateral costal branch usually describes a curve convex anteriorly. Bean (1905) states that the artery may reach the diaphragm, but no such extensive example was seen in the present series. The artery is accompanied by two veins between which it lies. The veins (Fig. 4) unite deep to the artery behind the first rib, then pass over the internal mammary artery to empty into the subclavian vein.

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ANESTHETIC EXPLOSIONS

Case Report

Maurice G. Buckles,* M.D., F.A.C.S., Columbus, Ohio

VERY little, if any, material on anesthetic explosions has been presented from the standpoint of the thoracic surgeon, and a very few cases of survivals in an anesthetic explosion have been reported, so that I feel it behooves one with this experience to record it, particularly with reference to management. It is quite probable that the pulmonary manifestations presented in this paper are primarily due to the blast of the explosion and simulate to a degree the blast noted in warfare, as well as in civilian life. There is little doubt that such changes in intrapulmonic pressure result in serious pulmonary conditions.

The author has no claims of being an anesthesiologist, and makes no pretense of discussing the various techniques of anesthesia, the chemistry of anesthetic agents, or the fine points of anesthesia as a whole. The following report is a case in which the author was called upon to aid in such an explosion, and was immediately available. An attempt is made to recite as accurately as possible the findings, the management, and the clinical course.

The occurrence of anesthetic explosions in the operating room is statistically uncommon. It is still too frequent, however, considering the danger to the patient, the operating room personnel, and the physical surroundings. Inasmuch as there has been no apparent decrease in the number of explosions, despite the numerous precautions recommended and undoubtedly practiced in connection with anesthesia,[1] it is important that the results and sequelae of such an explosion occurring to a patient under anesthesia be immediately recognized and handled expediently. This may tend to reduce the morbidity and mortality from such an accident.

The anesthetic explosion recently encountered resulted in severe hemorrhage into the paraorbital tissues; lacerations of the nasopharynx with profuse hemorrhage; prolonged hemorrhage from the tracheobronchial tree; production of a bilateral hemorrhagic pneumonia; spontaneous pneumothorax with an accompanying subcutaneous and mediastinal emphysema and pneumopericardia. The case report, which follows, presents the successful management; and also serves as a reminder that this dangerous accident still occurs.

CASE REPORT

H. S., a 56-year-old married white man, entered the hospital on Jan. 16, 1950, for the treatment of glaucoma. There was an associated diabetes mellitus, and hypertension—the blood pressure being 170/110.

On the fifth hospital day (Jan. 21, 1950) the patient was taken to surgery for an operation on the right eye. Anesthesia consisted of intravenous sodium pentothal with oxygen and nitrous oxide which was administered through a nasopharyngeal tube. Surgery consisted of cyclodiathermy to the right eye.

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At the conclusion of the operation there was a loud explosion, and the nasopharyngeal tube was blown out of the patient's nose, followed by profuse hemorrhage from the nose and mouth. His condition quickly became critical; the blood pressure dropped to 80/40; respirations were labored, and he became quite cyanotic. The author, who happened to be in the hospital, was called to see the patient. A 7 mm. bronchoscope was passed immediately with suction and aspiration of a large quantity of blood from the tracheobronchial tree. The patient resumed fairly normal respiration, and the cyanosis was relieved. The blood pressure gradually rose to 140/60. However, hemorrhage continued from the tracheobronchial tree, nasopharynx, and into the paraorbital tissues with marked swelling of the right cheek. An incision was made inside the mouth to decompress the swelling of the right cheek, and a pressure bandage was placed over the eyes. The nasopharynx was packed through the mouth to control the hemorrhage and also to prevent blood from running into the trachea; the anterior nares were then packed with gel foam. The bronchoscope was left in place for fifty minutes, but bleeding from the tracheobronchial tree continued, so an endotracheal tube was inserted to facilitate aspiration and to maintain an adequate airway. The patient was returned to his room in fair condition.

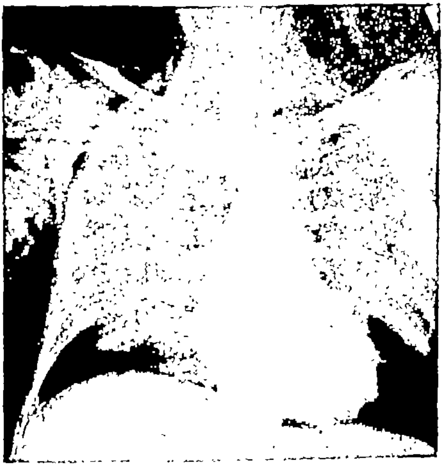

Fig. 1.—Portable chest roentgenogram following the explosion revealing subcutaneous emphysema, bilateral pulmonary infiltration, right pneumothorax, and mediastinal emphysema.

After returning to his room, the patient was placed on continuous oxygen, the tracheobronchial tree was aspirated frequently, and a blood transfusion was started. There were definite Cheyne-Stokes respirations for the first hour after being returned to his room, and he did not regain consciousness for about three hours. After receiving 500 c.c. of blood, the blood pressure was 100/70, the rectal temperature was 101.2° F., pulse 104, and respirations 20. Parenteral penicillin was started.

On the afternoon following the explosion, subcutaneous emphysema developed over the right anterior chest. Bleeding from the tracheobronchial tree continued, and about 600 c.c. of bloody fluid had been aspirated through the endotracheal tube. However, hemorrhage from the nasopharynx and into the paraorbital tissues was apparently controlled. A portable chest roentgenogram examination (Fig. 1) was made which revealed marked subcutaneous emphysema of the thoracic wall and cervical area; marked bilateral infiltration of the lung fields, although more extensive on the right side; and a small pneumothorax space on the right side with mediastinal emphysema.

The following morning (Jan. 22, 1950) the patient appeared in fairly good condition. The blood pressure was normal (preoperative level), the subcutaneous emphysema had not increased, there was no dyspnea, and bleeding from the tracheobronchial tree had apparently stopped. The packing from the nasopharynx and nose was removed; also the endotracheal tube. Following this, the patient was able to breathe freely, and was quite comfortable.

In the afternoon of the second postoperative day (Jan. 23, 1950) the patient developed progressive respiratory distress, the subcutaneous emphysema increased and extended to the head and neck, and the degree of pneumothorax was greater by physical examination. An intercostal catheter was introduced on the right side and attached to underwater drainage. There was an immediate escape of air with relief of the dyspnea, and the patient was again comfortable.

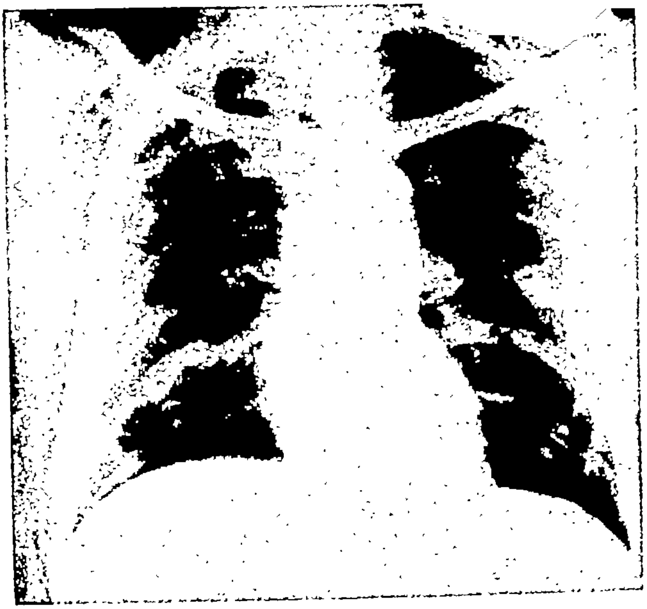

Fig. 2.—Chest roentgenogram examination on the fifth day showing clearing of the pulmonary infiltration, persistent subcutaneous and mediastinal emphysema, and evidence of pneumopericardia.

He rapidly improved, the subcutaneous emphysema diminished, breath sounds were good throughout both lung fields, and on Jan. 25, 1950, the intercostal catheter was removed. On Jan. 26, 1950, the temperature, pulse, and respirations were normal; a chest roentgenogram examination (Fig. 2) revealed marked clearing of the bilateral pulmonary infiltration, with persistence of the mediastinal and subcutaneous emphysema, and evidence of pneumopericardia.

In the evening of Jan. 26, 1950, the spontaneous pneumothorax recurred and was treated by repeated aspirations. By Jan. 28, 1950, the lung was reexpanded and his general condition was excellent. A chest roentgenogram examination on Feb. 2, 1950, revealed complete clearing of the lung fields and reexpansion of the right lung with some subcutaneous emphysema.

On the fourteenth day following the explosion, Feb. 4, 1950, the patient was discharged to the care of the ophthalmologist.

DISCUSSION

The exact mechanism of the anesthetic explosion in the case presented has not been ascertained; however, there are numerous reports concerning the explosions in the literature. The occurrence of such an accident should emphasize the importance of safety measures used to prevent such explosions, since prevention is the best method of treatment. Although it is stated that there has been no apparent reduction in the number of explosions despite the known safety precautions, it is perhaps questionable how conscientiously all of the safety measures are practiced in all operating rooms by all of the operating room personnel. This is especially an important question since all too often the personnel are divided into those "who have" and those "who have not" witnessed an explosion with disastrous results. It is almost a certainty that those "who have" will never forget it.

It is obvious that the prevention of such explosions is of prime importance not only for the safety of the patient and personnel, but also from medicolegal viewpoints. A disastrous explosion in absence of or in violation of recommended safety precautions might produce a weak argument in court or in answering the questions of the patient's relatives.[3]

The recommended safety precautions have been summarized by Cole[2] as follows: the role of the manufacturer of anesthesia apparatus includes the production of equipment which will reduce the possibility of ignition of combustible gases. Precautions to be taken by the anesthetist include: (a) the avoidance of combustible agents in the presence of obvious hazards, such as the cautery; (b) eliminating the practice of washing out patient and apparatus with oxygen following an anesthesia; (c) use of caution in spilling gas; (d) employment of the intercoupler or the maintaining of continuous contact by the anesthetist with patient and machine; (e) use of the rebreathing technique; (f) washing tubes, bags, and mask with water or calcium chloride solution; (g) elimination of unnecessary, sudden movements by the anesthetist, and of unnecessary adjustments of the mask. The duties of the hospital are: (a) the installation of safe lights, switches, and electrical wiring; (b) the maintenance of a high humidity in the operating room; (c) prohibition of wool, silk, and ordinary rubber; (d) banning of visitors from the area surrounding the anesthetic apparatus; (e) banning of open flame, cigarettes, etc., from the operating room; and (f) the construction of grounded, conductive floors.

SUMMARY

1. A case report of an anesthetic explosion has been presented to demonstrate a method of handling such an accident, and also as a reminder of the constant danger involved in handling anesthetic gases.

2. The case involved was marked by severe hemorrhage into the paraorbital tissues; hemorrhage from lacerations of the nasopharynx; prolonged hemorrhage from the tracheobronchial tree; production of a bilateral hemorrhagic pneumonia; a spontaneous pneumothorax with subcutaneous and mediastinal emphysema, and pneumopericardia.

3. The importance of recognizing the potential explosive hazard of all inhalation anesthetics, and using accepted precautionary measures to prevent such accidents is emphasized.

4. The accepted safety measures are reviewed.

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THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

THIRTY-FIRST ANNUAL MEETING

APRIL 16, 17 AND 18, 1951, CHALFONTE-HADDON HALL
ATLANTIC CITY, N. J.

PROGRAM

Monday Morning, April 16, 1951

9:00 A.M. BUSINESS MEETING

9:30 A.M. SCIENTIFIC SESSION

1. **Progressive Changes of Pulmonary Function After Pneumonectomy: The Influence of Thoracoplasty, Pneumothorax, Oleothorax and Plastic Sponge Plombage on the Pneumonectomized Side**
   Edward A. Gaensler (by invitation) and John W. Strieder, Boston, Mass.
2. **Pulmonary Function in Traumatic Hemothorax Treated by Decortication**
   James H. Forsee and Major Stephan L. Kylar (by invitation), Denver, Colo.
3. **Late Changes in Ventilatory Function Following Thoracoplasty**
   S. R. Powers, Jr. (by invitation), and A. Himmelstein, New York, N. Y.
4. **Lung Function Studies in Poudrage Treatment of Recurrent Spontaneous Pneumothorax**
   Lt. Col. John Paul (by invitation), Edward J. Beattie, Jr., and Brian Blades, Washington, D. C.
5. **Effect of Position on the Rise in Carbon Dioxide Tension During Thoracic Surgery and the Effect of Artificial Ventilation in the Several Positions**
   Henry K. Beecher, Thomas J. Quinn, Jr., John P. Bunker, and Genesio L. D'Alessandro (all by invitation), Boston, Mass.
6. **Mucoid Impaction of the Bronchi**
   Robert R. Shaw, Dallas, Texas

Monday Afternoon, April 16, 1951

2:00 P.M. SCIENTIFIC SESSION

7. **Changes in Urine and Serum Electrolytes and Plasma Volume, After Major Intrathoracic Operations**
   Robert K. Finley, Jr. (by invitation), John Y. Templeton, III (by invitation), Robert H. Holland (by invitation) and John H. Gibbon, Philadelphia, Pa.
8. **The Treatment of Cardiospasm: Analysis of a Twelve-Year Experience**
   Arthur M. Olsen (by invitation), Stuart W. Farrington, Herman J. Moersch, and Howard A. Andersen (by invitation), Rochester, Minn.
9. **Considerations as to Etiology and Treatment of Achalasia of the Esophagus**
   Earle B. Kay, Cleveland, Ohio
10. **Complications and Surgical Treatment of Hiatus Hernia and Short Esophagus**
   Donald B. Effler, Cleveland, Ohio
11. **Formation of a Temporary External Esophageal Fistula Over a T-Tube for Stenosing Esophagitis**
   Philip Thorek (by invitation), Chicago, Ill.
12. **Endoscopic Sponge Biopsy**
   Max G. Carter (by invitation), Clinton A. Piper (by invitation), and Robert Nesbitt (by invitation), New Haven, Conn.

Tuesday Morning, April 17, 1951

9:00 A.M. SCIENTIFIC SESSION

13. **Thoracoplasty Combined With Resection for Pulmonary Tuberculosis**
William S. Conklin and Jerome T. Grismer (by invitation), Portland, Ore.

14. **Results in 278 Patients Who Had Modern Type of Thoracoplasty for Tuberculosis**
William M. Lees, Stephen C. H. Yang (by invitation), Michael Papoulakos (by invitation), Jan K. Bosch (by invitation), John Alexander, and Angel Larralde (by invitation), Ann Arbor, Mich.

15. **Simultaneous Decortication and Resection in Ineffective Pneumothorax**
Arnold O. Riley (by invitation) and Victor Kaunitz (by invitation), Mount Morris, N. Y.

16. **Decortication Preceding Thoracoplasty for Eradication of Long Standing (Up to Six Years) Chronic Tuberculous and Mixed Infection Empyema**
F. Douglas Ackman, Montreal, Canada

17. **Simple Excision in the Treatment of Pulmonary Tuberculosis**
Bernard J. Ryan (by invitation), Edgar M. Medlar (by invitation), Sunmount, N. Y., and Edward S. Welles, Saranac Lake, N. Y.

18. **Subscapular Paraffin Pack as a Supplement to Thoracoplasty as a Collapse Procedure**
W. E. Adams, William M. Lees, and James M. Fritz (by invitation), Chicago, Ill.

Tuesday Afternoon, April 17, 1951

2:00 P.M. EXECUTIVE SESSION

3:00 P.M. SCIENTIFIC SESSION

Address of the President—Alfred Blalock, Baltimore, Md.

19. **The Pathogenesis of Bronchopleural Fistulas Following Excisional Therapy for Pulmonary Tuberculosis**
G. N. Stemmermann (by invitation), C. Daniels (by invitation), and O. Auerbach, New York, N. Y.

20. **Bronchiectasis: A Comparative Study of Tuberculous and Nontuberculous Pyogenic Suppurative Disease**
Joseph Gordon, Ray Brook, N. Y., and Philip C. Pratt (by invitation), Saranac Lake, N. Y.

21. **The Prognosis of Residual Bronchiectasis After Incomplete Resection**
George P. Rosemond, W. Emory Burnett, and Joan-Humphrey Long (by invitation), Philadelphia, Pa.

7:00 P.M. COCKTAIL PARTY—CHALFONTE-HADDON HALL

8:00 P.M. BANQUET—CHALFONTE-HADDON HALL
DANCING

Wednesday Morning, April 18, 1951

9:00 A.M. SCIENTIFIC SESSION

22. **Radical Pneumonectomy**
William G. Cahan (by invitation), William L. Watson, and John L. Pool, New York, N. Y.

23. **A Preliminary Report of 26 Cases of Pulmonary Resection With Pleurectomy**
Bert H. Cotton and John R. F. Penido (by invitation), Beverly Hills, Calif.

24. **The Management of the Patient With Inoperable Cancer of the Chest**
Richard H. Meade, Jr. and Richard A. Rasmussen, Grand Rapids, Mich.

25. **Radio Active Isotopes in the Palliative Management of Carcinomatosis of the Pleura**
Edward M. Kent and Campbell Moses (by invitation), Pittsburgh, Pa.

26. **Identification of Regional Lymph Nodes in the Treatment of Bronchiogenic Carcinoma**
    Joseph A. Weinberg, Long Beach, Calif.
27. **Pneumonectomy for Severe Irradiation Damage of Lung**
    Martin Bergmann (by invitation) and Evarts A. Graham, St. Louis, Mo.

Wednesday Afternoon, April 18, 1951

2:00 P.M. SCIENTIFIC SESSION

28. **Reconstructive Surgery of the Trachea and Bronchi: Late Results With Dermal Grafts**
    Paul W. Gebauer, Honolulu, Hawaii
29. **Myxochondroma of the Trachea: A Case Report**
    Byron H. Evans, Los Angeles, Calif.
30. **Analysis of Twenty-Four Cases of Acute Cardiac Arrest**
    J. L. Ehrenhaft, D. W. Eastwood (by invitation), and L. E. Morris (by invitation), Iowa City, Iowa.
31. **Constrictive Pericarditis and Constrictive Pleuritis Treated by Pericardectomy and Pulmonary Decortication**
    Richard H. Overholt, C. Sidney Burwell (by invitation), John W. Woodbury (by invitation), and James H. Walker (by invitation), Boston, Mass.
32. **The Direct (Brock) Relief of Pulmonary Stenosis in Tetralogy of Fallot**
    Robert P. Glover, Charles P. Bailey, and Thomas J. E. O'Neill (by invitation), Philadelphia, Pa.
33. **Treatment of Coronary Artery Insufficiency by Implantation of the Internal Mammary Artery into the Left Ventricular Myocardium**
    Arthur Vineberg, Montreal, Canada

PAPERS TO BE READ BY TITLE

1. **Further Discussion of the Right Thoracic Approach to Esophageal Lesions**
    R. K. Hollingsworth, Martinsburg, W. Va.
2. **Congenital Cystic Disease of Lung Associated With Anomalous Arteries**
    Frederick G. Kergin, Toronto, Canada
3. **Transpleural Bronchotomy**
    Alfred Goldman, Beverly Hills, Calif.

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Original Communications

LUNG FUNCTION STUDIES

VII. THE EFFECT OF PNEUMONECTOMY ON RESPIRATORY DEAD SPACE

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WITH pneumonectomy, part of the bronchial tree is removed. However, there is disagreement about the effect of pneumonectomy on the volume of the respiratory dead space. It has been reported that, after pneumonectomy, the dead space is enlarged;[1, 2] or that it is decreased.[3, 4] The former situation, if present, would be a considerable factor contributing to respiratory inefficiency.

The development by Lilly and Hervey of the N_2 meter, for continuous analysis of the N_2 concentration of respired gases, has made possible the ready measurement of respiratory dead space both of normal subjects[5] and of many patients with abnormally uneven alveolar ventilation.[6] This measurement of respiratory dead space represents the volume of inspired gas which remains in the conducting airway and does not contribute to dilution of alveolar gas. It probably approximates the so-called anatomic dead space, and is practically independent of alveolar ventilation-perfusion relationships, which affect other types of "dead space" measurement using oxygen or carbon dioxide values.[7]

Measurements of the respiratory dead space in patients before and after pneumonectomy are presented; the postoperative values were similar to those of healthy control subjects. Other studies made after pneumonectomy suggest that the large "dead space" values reported by Birath were caused by abnormally uneven alveolar ventilation, and not by an actual enlargement of the dead space.

METHODS

Eight patients (7 males and 1 female) were studied after pneumonectomy for pulmonary neoplasm; seven were *also* studied a few days preoperatively. Eleven elderly (53 to 71 years) male hospital patients without clinical or fluoro-

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scopic evidence of pulmonary disease served as controls. All subjects were examined semi-reclining in bed, with the head and trunk elevated 50 to 60 degrees. Subjects were not in basal condition but had rested in bed at least ten minutes before measurements were made.

The method has been described previously.[5, 6] In brief, it consists of continuous analysis and photographic recording of N_2 concentration and volume flow of gas expired during and after the change from breathing room air to breathing 99.6 per cent oxygen. Although the dead space of most young normal subjects can be measured directly on the photographic records,[5] it was necessary here to replot two or three successive expirations (occurring during the first thirty seconds of O_2 inhalation) as curves of N_2 concentration versus expired volume.[6]

Dead space measurements made on different days (before and after pneumonectomy) might differ because of error in the method and also because of day-to-day variation, quite independently of the operation. Therefore duplicate measurements were made on six healthy subjects on each of two different days. By analysis of variance[8, 9] of these measurements and of the duplicate measurements on the patients, it was found that (a) the error of the method was similar in the patients, the six healthy subjects noted above, and in forty-five healthy subjects studied previously,[5] and (b) a difference of 42 ml. or more between the means of duplicate pre- and postoperative measurements, made on different days, as given in Table I, is significant and may reasonably be ascribed to the operation.

The method requires the inhalation of O_2, which, over a five minute period, usually causes a reduction of tidal volume in chronically anoxemic patients, and yields a larger dead space/tidal volume fraction than if the average tidal volume observed during a five minute period of breathing air is used. The dead

TABLE I. RESPIRATORY DEAD SPACE BEFORE AND AFTER PNEUMONECTOMY,* AND IN NORMAL ELDERLY MALES

	SEX	AGE YRS.	HT. CM.	RESP. D. S. ML.		TIDAL VOL. ML.	DS/TV x 100 %	INTERVAL	PNEUMONECTOMY
Normal†	M	59.6	166	150		623	24.9	----	----
Mean ± S.D.				±31		±149	±6.1		
				pre	postoperative				
Na	M	60	171	187	133	494	27.0	13 days	R
Ws	M	59	175	154	144	452	31.8	13 days	L
Fr	M	55	173	203	140	582	24.1	12 days	L
Wh	M	65	161	--	130	386	33.6	3 mos.	R
Gn	M	54	178	205	158	576	27.4	12 days	R
Cy	M	68	165	178	190	906	21.0	5 mos.	R
Ry	M	52	167	202	145	670	21.6	9 mos.	L
Mn	F	56	165	137	125	393	31.8	15 days	R
Mean (males)	–	59	169	188	149	581	26.6	----	--

*All male patients had carcinoma, *Mn* had mesothelioma; in addition, *Cy* had tuberculosis, silicosis and emphysema and *Ry* had emphysema.

†These 11 subjects were reported elsewhere.[4]

space/tidal volume fractions given (Table I) were calculated from average tidal volumes during the breathing of air, measured with a recording 120-liter spirometer. All volumes are corrected to BTPS.

RESULTS

Data for patients and control subjects are given in Table I. Preoperatively, the dead space values in men, 154 to 205 ml., fell in the upper range of normal values, 150 to 212 ml. (average plus 2 S.D.). The mean value of 188 ml. for the male patients was significantly ($p = 0.02$) greater than the mean control value of 150 millimeters.

Postoperatively there were no significant changes in the dead space values for two men and one woman. The lack of change in the woman may have been due to a dead space reduction also existing preoperatively, caused by compression stenosis of the right bronchial tree, including the right main bronchus, from the large tumor occupying about two-thirds of the right hemithorax. Four male subjects, none of whom had obstruction of main bronchi preoperatively, had significant postoperative reductions of about 50 millimeters.

The mean postoperative values of male patients for dead space and dead space/tidal volume fraction were similar to the values for the healthy males.

DISCUSSION

The explanation of the enlargement of the mean preoperative dead space value is not entirely clear. In the two cases with pulmonary emphysema, the values of 178 and 202 ml. probably reflect the over-all state of pulmonary hyperinflation exhibited during quiet breathing by such patients. A similar explanation may apply to some extent to the other cases; enlargement of the functional residual capacity has been reported in similar patients.[1]

The mean postoperative values for dead space and dead space/tidal volume fraction reported here are significantly smaller than the respective mean values of 236 ml. and 48.7 per cent reported by Birath,[1] which, with other similar findings,[2] were taken to indicate a probably actual enlargement of the dead space resulting from postoperative pulmonary distention. The difference very likely results from the methods used. Birath examined his patients in a sitting position, in which the respiratory dead space is about 25 ml. greater than when semi-reclining.[10] Probably even more important was the presence of uneven pulmonary ventilation in his subjects. In five of our patients studied postoperatively it was possible to make calculations similar to those used by Birath, since the alveolar (end-tidal) N_2 concentrations and tidal volume were measured continuously during the first two minutes of O_2 inhalation, and functional residual capacity was also measured. Four of these patients were found to have abnormally uneven ventilation by the method described elsewhere,[11] and their calculated "dead space" values were all larger, average 295 ml., than the direct measurements, average 152 ml.; the values were similar in one patient (Mn) with normal ventilation uniformity. The reasons for the discrepancy between the calculated and directly measured values follow. In Birath's calculations, based on rebreathing a gas mixture containing H_2, and in ours, which were based on the pulmonary N_2 elimination equations of Darling and associates,[12] it is assumed that the lungs are ventilated uniformly, and that the ventilatory process can be expressed by an exponential equation, stating that the alveolar gas concentration is changed by a certain fraction on each successive inspiration of the "foreign gas." For example, if 300 ml. of O_2 (tidal volume of 450 ml. minus 150 ml. dead space) are added to the functional residual capacity of 2700 ml., containing 80 per cent N_2, the N_2 concentration will be reduced to $\frac{2700}{3000}$ or 0.9 times 80 per cent; the next breath will reduce the concentration to 0.9 times 72 per cent, etc. Experimentally one obtains the value of the fraction, and, knowing tidal volume and functional residual capacity, can solve for the dead space. However, with uneven ventilation, the hyperventilated areas of the lungs approach equilibrium with the inspired gas more rapidly (smaller value of the fraction) than do the relatively hypoventilated areas. Therefore, early in the procedure the expired gas will reflect the rapid rate of change in the hyperventilated areas more than it does later, when these areas have reached equilibrium and the expired gas is manifesting the slower rate of change in the hypoventilated areas. Such a multiphasic ventilation process cannot be expressed accurately by equations containing only one fractional value expressing the rate of change. One can calculate dead space using one fractional value, but the result varies with the portion of the equilibration curve from which the fraction was obtained. The rapid change early in the procedure yields a very small value for dead space, the slower change later in the procedure yields an excessively large value for dead space. Birath's calculations were apparently based principally on a measurement made after a one-minute breathing period; similar calculations applied to the one-minute point on the alveolar N_2 elimination curves gave the artificially large "dead space" values noted above.

The effect of pneumonectomy on the volume of the respiratory dead space is not easily anticipated; there appear to be various changes which could either diminish or enlarge it. The dead space, as measured here, represents the volume of inspired gas which remains in the conducting airway and does not contribute to dilution of alveolar gas. This volume is affected by the anatomic volume of the conducting airway and also by the extent of gaseous diffusion occurring between terminal bronchioles and alveolar spaces; prolongation of inspiratory time results in a smaller dead space, presumably because the peripheral boundary of pure inspired gas recedes up the bronchial tree.[5]

Partial resection of the bronchial tree would tend to reduce the dead space. Rohrer[13] estimated that about 45 per cent of the volume of the conducting airway was below the tracheal bifurcation. One might therefore expect a 20 to 25 per cent reduction with pneumonectomy; the mean change observed postoperatively was a 17 per cent reduction. However, part of the remaining airway is intrathoracic, and probably participates in any postoperative distention of the remaining lung. Also, the dead space might possibly be enlarged by a reduction in the extent of gaseous diffusion occurring between terminal bronchioles and alveolar spaces. This could result from shortened diffusion time accompanying a postoperative increase of respiratory frequency (an average increase of 2.4 respirations per minute was noted in the patients above); a change in what may loosely be called diffusion surface might result when a total alveolar volume considerably greater than half normal is connected to bronchioles presumably only half normal in number.

A postoperative dead space volume of 150 ml., although similar to normal values, is in one sense a respiratory handicap. Normally only about 75 ml. of the tidal volume developed by each lung is not effective in ventilating the alveoli; thus the "wasted effort" of the remaining lung is doubled. This, in addition to other departures from the normal thoracic mechanics, represents an encroachment upon the reserve of the bellows or ventilatory mechanisms, and may contribute to the postoperative tendency to experience dyspnea. However, in another sense, a postoperative bronchial distention might be favorable. After pneumonectomy, the patient is obliged to force through one set of bronchial tubes about the same respiratory minute volume which is normally forced through two sets of tubes. The increase in respiratory effort needed for this would probably be reduced by bronchial distention in the remaining lung.

SUMMARY

The respiratory dead space was measured before and after pneumonectomy on patients breathing quietly in the semi-reclining position. Postoperatively, three subjects showed no change, and four male patients showed significant reductions. The mean postoperative values for dead space and dead space/tidal volume fraction were similar to control values for elderly men. Factors affecting the volume and measurement of respiratory dead space after pneumonectomy are discussed.

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OBSTRUCTIVE EMPHYSEMA WITH A DEFECT OF THE ANTERIOR MEDIASTINUM

REPORT OF A CASE

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OPENINGS in the anterior mediastinum with direct communication between two pleural cavities are very rare congenital abnormalities. Kupfer[1] studied mediastinal lesions in 6,000 autopsies and did not observe such an anomaly. In 1939, Ochsner, DeBakey, and Murray[2] reported a case of congenital absence of the anterior mediastinum visualized at operation for repair of a diaphragmatic hernia in an 18-month-old child. The entire anterior mediastinum was absent, and the right hemithorax could be visualized from the left thoracic wound. The pericardium and great vessels lacked their normal attachment to the posterior aspect of the sternum. The diaphragmatic hernia was repaired, but no repair of the mediastinum was carried out. The infant made an uneventful recovery.

In their report, a review of the literature revealed only nine other reports of human subjects suspected of having a passageway connecting the two pleural spaces. However, the report was the first clearly to describe the anatomic findings, which without a doubt, represented a congenital abnormality of the mediastinum.

Since that time a review of the literature reveals only one other case of proved congenital absence of the anterior mediastinum. In 1945, Gross and Lewis[3] reported an absent anterior mediastinum in a 4-year-old child who was operated upon for obstructive emphysema of the right upper lobe caused by absent cartilaginous rings in the right upper lobe bronchus. A right upper lobectomy was carried out, and the anterior mediastinum reconstructed by suturing the pericardium to the sternal periosteum with two rows of interrupted silk sutures. This was the first report of a successful surgical repair of the anterior mediastinum.

In view of the rarity of the defect, one of us (J. E. L.) feels rather fortunate in having been able to collaborate with Dr. Gross in reporting the previously proved case in 1945 and to add yet another case of anterior mediastinal defect associated with obstructive emphysema of the right middle lobe of the lung.

CASE REPORT

A 4-month-old white male infant was admitted to the Children's Memorial Hospital on the service of Dr. Stanley Gibson, June 1, 1950, with a history of dyspnea and cyanosis when crying noted since 6 weeks of age.

From the Surgical Service of the Children's Memorial Hospital, Chicago, Ill.

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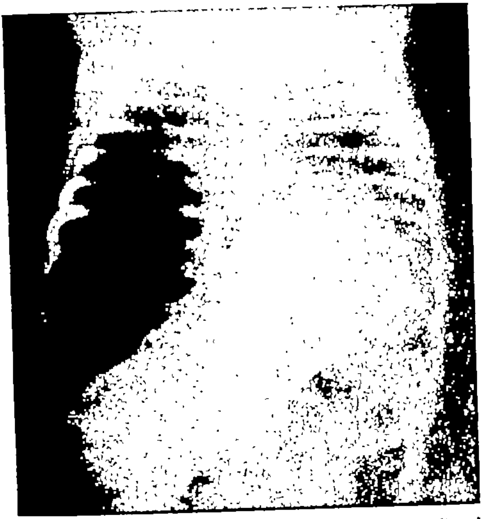

Fig. 1.—Anteroposterior roentgenogram shows the heart pushed to the left, collapse of the right lower lobe, partial collapse of the right upper lobe, and marked emphysema of the middle lobe.

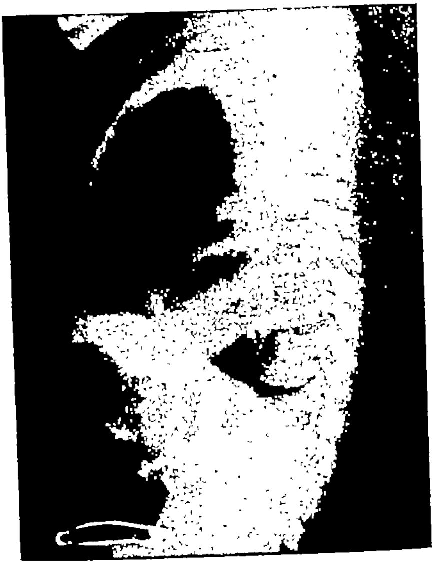

Fig. 2.—The lateral roentgenogram shows a large retrosternal radiotranslucent area. The heart is pushed posteriorly.

The infant had an uncomplicated spontaneous birth following a normal pregnancy. He weighed 5 pounds, 5 ounces at birth, and progressed normally in weight gain and development without associated symptoms until 6 weeks of age. At this time it was noted that the infant became blue and dyspneic when crying. However, there were no other symptoms, and the infant developed normally until six weeks before entry to the hospital. At this time he developed paroxysms of cough accompanied by crying, cyanosis, and rapid respirations. The attacks occurred approximately every half hour, and lasted from five to ten minutes. Associated with these attacks, he had difficulty in eating and lost weight. Feeding seemed to initiate difficulty in breathing and produced crying and cyanosis. Consequently he was brought to the hospital for evaluation and study.

Physical examination on admission revealed a rectal temperature of 104° F., a rapid pulse, and rapid respirations. The infant was well developed and well nourished, but slightly cyanotic. Respirations were grunting in character, punctuated by a dry hacking cough. Physical findings were essentially normal except for the chest examination. The anteroposterior diameter of the chest appeared prominent. The heart was shifted to the left, and the heart sounds were distant with no murmurs. There was a hyperresonance to percussion over the right anterior chest and over the cardiac area. Breath sounds were distant on the right side of the chest and normal on the left. No râles were heard.

Laboratory examination revealed a white blood count of 15,800, with 32 per cent polymorphonuclear leucocytes, 60 per cent lymphocytes, 5 per cent monocytes, 1 per cent eosinophiles, and 2 per cent stab forms; a hemoglobin of 10.4 grams per cent, and a red blood count of 4.42 million. The urine was normal.

Roentgenograms of the chest revealed emphysema of the right middle lobe with atelectasis of the right upper and lower lobes. The heart was shifted to the left. In the lateral view there was a clearly demarcated globular area of emphysema lying anterior to the heart beneath the sternum. The diagnostic impression was obstructive emphysema involving the right middle lobe of the lung (Figs. 1 and 2).

The infant's grave condition precluded further studies, and thoracotomy was carried out as an emergency procedure. The infant was anesthetized with endotracheal cyclopropane. A polyethylene tube was inserted in the right ankle vein for the administration of blood.

The infant's condition was very precarious under anesthesia because of the poor exchange of gases in the small amount of functioning lung, and thoracotomy was rapidly carried out through the fourth interspace. The large emphysematous middle lobe delivered itself partially through the operative wound, and the pulmonary difficulties were sufficiently relieved to allow deliberate procedure with the operation. Inspection revealed a large emphysematous middle lobe occupying the right chest cavity and collapsing the right upper and lower lobes. On withdrawing the remainder of the middle lobe from the chest cavity, it could be seen that the middle lobe extended across the midline anterior to the pericardium into the left hemithorax through a large anterior mediastinal defect. The left lung could be visualized without difficulty through the defect in the anterior mediastinum. Consequently a right middle lobectomy was carried out. The vessels were dissected out individually, ligated, and divided. The bronchus which appeared grossly normal was clamped with a toothed ductus clamp, divided and sutured with interrupted No. 00000 silk sutures. Pleura was then sutured over the stump of the bronchus. The question of closure of the anterior mediastinum then arose. It was felt desirable, but inadvisable, in view of the precarious state of the patient, the technical difficulty from the posterolateral approach, and the increased anteroposterior diameter of the chest which would have contributed marked tension to the suture line.

Consequently, the chest was closed in layers with a catheter inserted into the pleural cavity through the seventh interspace for drainage.

At the close of the operation the infant's condition seemed good; however, as the intratracheal catheter was removed, respirations ceased. The catheter was quickly reinserted and the patient revived with artificial respiration and oxygen. This sequence of events took place three more times before normal respiration was reestablished. The infant was returned to his crib approximately one hour after surgery in good condition. His postoperative course was excellent (Fig. 3). Just prior to discharge on the tenth postoperative day, roentgenograms revealed that the heart had shifted back to its normal position, and there was complete expansion of the right upper and lower lobes (Fig. 4). No evidence of recurrent emphysema or residual atelectasis was seen. The infant was discharged without symptoms, and when seen recently at the age of 7 months was in excellent condition.

Pathologic review of the lobe revealed no cause for the emphysema. The bronchus, bronchi, and bronchioles appeared patent and normal. The lung presented a typical picture of emphysema. The main bronchus was sectioned, and revealed no cartilaginous ring defects or obstruction.

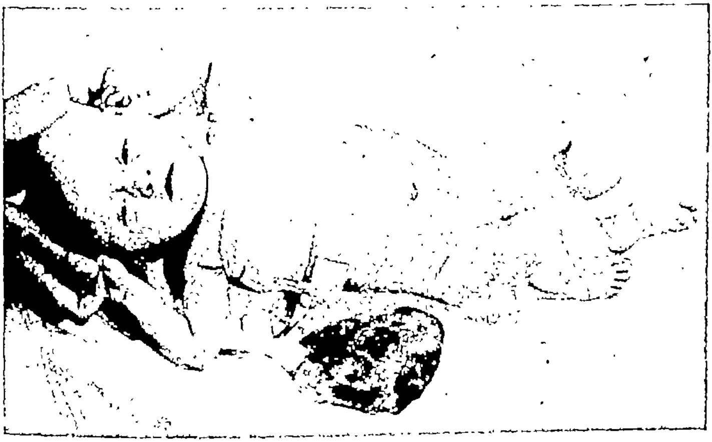

Fig. 1.—Photograph of infant, forty-eight hours after surgery, with the removed middle lobe. Note the size of this lobe compared with that of the baby's chest.

DISCUSSION

This case is significant because it is only the third proved published case of complete absence of the anterior mediastinum undoubtedly of congenital origin. However, of further interest is the possible relationship of the absent anterior mediastinum to the development of the obstructive emphysema. No definite abnormality of the right middle bronchus could be determined either grossly at the time of operation or microscopically in the pathologic specimen. Unfortunately, the condition of the infant prevented bronchoscopy and bronchograms, and an obstruction at the origin of the right middle bronchus may have been overlooked. However, the gradual development of symptoms with the initial symptoms at 6 weeks of age, the presence of the right middle lobe herniated through the wide congenital defect of the mediastinum, and the apparent absence of abnormalities of the bronchus to account for the emphysema cause one to suspect that the anterior mediastinal defect may not have been purely coincidental, but may have contributed to the development of the emphysema.

The anterior mediastinum represents the midline partition persisting after expansion of the parietal pleurae ceases. Before birth the lungs are relatively small and compact. They occupy only the dorsal portions of the thorax, while the anterior mediastinum is quite extensive and is very broad. With the onset of breathing at birth, the lungs distend with air, assume a more anterior position, and press and narrow the anterior mediastinum. The anterior and central position of the middle lobe make it more vulnerable to herniation through a mediastinal defect. Consequently, in this case we feel that it is highly probable that the herniation of the middle lobe through the anterior mediastinal defect resulted in kinking of the middle lobe bronchus with subsequent gradual development of the massive obstructive emphysema. Perhaps one should add this congenital abnormality to the possible etiological agents in obstructive emphysema. The striking lateral films of the chest in this case and the case reported by Gross and Lewis are very suggestive of anterior mediastinal defect, and should justify a presumptive diagnosis preoperatively.

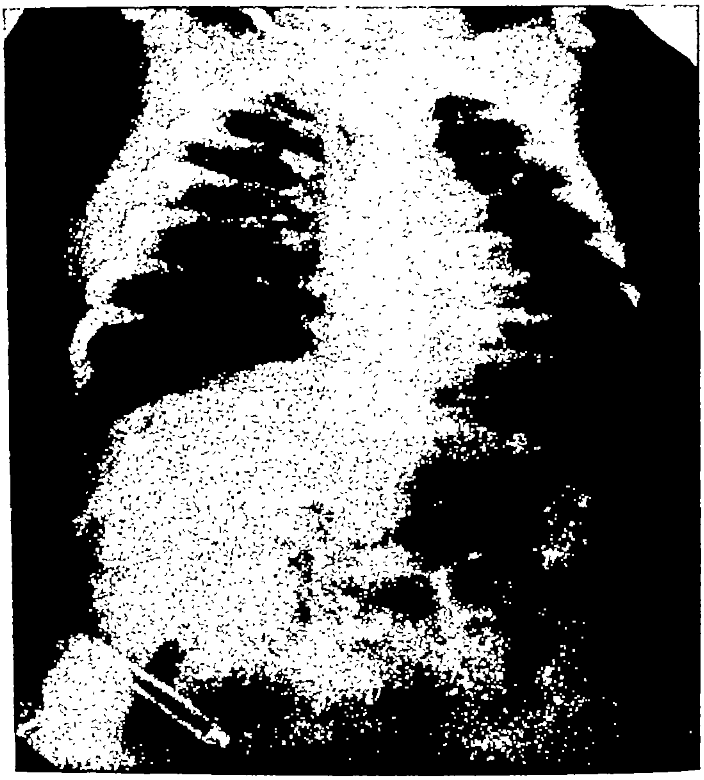

Fig. 4.—Anteroposterior roentgenogram on the tenth postoperative day showing the heart in normal position with complete expansion of the right upper and lower lobes.

Should such a mediastinal defect be closed? There is insufficient recorded experience to furnish an answer. It would appear that if there were any coexistent disease or malformation which might unbalance the pressure in the two sides of the chest, the defect should be closed. However, if a defect of the mediastinum were an incidental finding as in Ochsner's case, it would seem better judgment to leave it as found. It is not a simple matter to close a large mediastinal defect. Furthermore, untoward complications conceivably might arise from drawing the pericardial sac with many fine interrupted silk stitches to the periosteum of the posterior surface of the sternum. The mediastinal defect in our patient was not closed because the condition of the patient did not warrant extension of the operating time. It did not seem likely after the emphysematous middle lobe had been removed that there was much chance of further complications from this defect.

SUMMARY

A case report is made of a 4-month-old male infant who had a large anterior mediastinal defect with free interpleural communication and obstructive emphysema of the middle lobe producing severe respiratory symptoms.

The possible role of the anterior mediastinal defect as an etiological agent in the development of the obstructive emphysema in this case is discussed.

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CAVERNOUS HEMANGIOMA OF THE THORACIC WALL ASSOCIATED WITH CLINICAL EVIDENCE OF ARTERIOVENOUS FISTULAS

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WE ARE reporting data on a patient recently observed at the Mayo Clinic who had a large cavernous hemangioma associated with clinical evidence of arteriovenous fistulas involving the seventh, eighth, and ninth intercostal vessels of the left posterior thoracic wall. Resection of the lesion was successfully accomplished.

In a review of the literature in English since 1935, we found no case of cavernous hemangioma associated with clinical evidence of arteriovenous fistula involving the intercostal vessels, in which surgical treatment had been carried out. Cavernous hemangiomas of the thoracic wall which are not associated with clinical evidence of arteriovenous fistula and which require surgical treatment are uncommon enough in themselves. Such lesions involving the bones of the thoracic wall have been reported by Bucy and Capp,[1] Mallory,[2] and Dorner and Marcy.[3] Excision of the involved bone with recovery has been the rule, although some of these patients were successfully treated with roentgen therapy. Shallow and his co-workers,[4] in a review of 335 cases of primary hemangiomatous tumors of skeletal muscle, found thirty-nine involving muscles of the thoracic wall, of which four involved intercostal muscles. Of the entire series, a bruit was heard in 1.8 per cent, and a thrill was noted in 0.6 per cent. At least one of the hemangiomas involving the thoracic wall was associated with both a thrill and a bruit. This was the case of Serra, Lavenia, and Frega,[5] in which the lesion was said to involve the muscles of the right hemithorax. Excision was carried out in this case, with recovery.

Elkin and Cooper[6] reported three large hemangiomas of the thoracic wall, two of which were completely excised. A bruit was not specifically associated in the report with any of these, although in three of the nineteen extensive hemangiomas from all parts of the body with which this report dealt, pulsations were present, and in two a bruit was audible, and was interpreted as evidence of large arteriovenous communications.

During the fifteen-year period from 1935 through 1949, fifteen cases of cavernous hemangioma, in which surgical treatment was carried out, were seen at the clinic. In none of these cases was there clinical evidence of arteriovenous fistulas, as indicated by the findings of a thrill or a bruit, nor was there any roentgenographic evidence of erosion of the ribs. Three of the hemangiomas possibly involved intercostal vessels, but since the lesions were only partially excised, their true extent could not be determined.

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Several cases have been reported in which the presumptive diagnosis of arteriovenous fistula has been made, but definitive treatment was not undertaken. The earliest such case which we could find in the literature in English was that of Langmead,[7] reported in 1912. His patient was a woman aged 55 years with tortuous pulsating vessels over the posterior aspect of the thorax, associated with a thrill and a loud systolic bruit, the pitch of which could be altered by pressure. A rounded circumscribed shadow between the third and fourth ribs posteriorly was seen on the roentgenogram of the thorax. This was thought to be an intercostal aneurysm, but a cirsoid aneurysm was also considered. Pathologic proof was not available.

Hench and Horton[8] described bruits in two female patients, in both of whom a continuous murmur was heard in the seventh interspace on the left, just beneath the lower tip of the scapula, posteriorly. There were no other local signs. Pressure over the bruit did not cause slowing of the heart rate. In one of these patients, roentgenographic evidence of scalloping of the inferior borders of the seventh and eighth ribs on the left was seen. The clinical diagnosis in each case was arteriovenous fistula of the intercostal vessels. No attempt was made to treat the condition.

Maliner[9] reported the case of a boy aged 12 years with an epigastric tumor mass "like a bundle of worms" over which a systolic murmur could be heard, which could be made to disappear with firm pressure. The clinical diagnosis was cavernous hemangioma. There was no roentgenographic evidence of involvement of bone. Surgical treatment was not undertaken.

One case has been encountered at the clinic in the past fifteen years, in which an extracardiac bruit suggestive of an arteriovenous fistula involving vessels of the thoracic wall was heard. The patient was a woman aged 36 years, in whom a to-and-fro machinery murmur was observed over the xiphisternal area, extending over the precordium and left axilla, and posteriorly along the medial border of the left scapula. The results of roentgenographic study were negative, and cardiac catheterization did not reveal any abnormality of the heart or great vessels. There were no associated symptoms, and treatment was not felt to be necessary.

We were able to find two cases of posttraumatic arteriovenous fistula involving vessels of the thoracic wall. Shumacker and Carter[10] reported one among 354 cases of traumatic arteriovenous fistulas and arterial aneurysms. In this case, the fistula was between the internal mammary artery and the innominate vein. More recently, Elkin[11] reported the case of a female physician aged 39 years in whom a traumatic arteriovenous aneurysm of the vessels of the right side of the diaphragm was successfully excised. This patient had previously been considered to have an arteriovenous fistula of the ninth intercostal vessels, of more than twenty years' duration, following repeated thoracenteses for pleural effusion. There was a harsh machinery-like bruit with systolic accentuation in this area, which could not be obliterated by pressure. No erosion of the underlying ribs could be seen on roentgenographic examination.

A congenital arteriovenous fistula involving the left pectoralis major muscle was recently reported to have been successfully resected by Maier and Stout.[12] This lesion occurred in a woman aged 30 years in whom a continuous murmur in the precordial region had led to a previous diagnosis of patent ductus arteriosus. It was not until a mass underlying the left breast became palpable that the diagnosis of arteriovenous fistula of the thoracic wall was made. This mass was slowly expanding, and a continuous bruit with systolic accentuation was heard over it. A thrill which could be diminished by firm pressure was also observed. These authors also mentioned having observed a patient with arteriovenous fistulas, one in the thoracic wall, and the other in the opposite lung, which was giving rise to hemoptysis.

CASE REPORT

A white fireman, aged 40 years, was first seen at the Mayo Clinic on July 10, 1950, complaining of a tumor mass of the posterior left portion of the thorax. The family history was noncontributory. The patient had been well all his life except for a traumatic amputation of the terminal phalanx of the left fifth finger. In February, 1948, while opening a fire hydrant, the patient felt something ''give'' in the region of the posterior axillary line of the left lateral portion of the thorax. There was pain in the area for two weeks. Treatment was strapping of the thorax. Continuous soreness was felt in this area until three or four months later, when a small mass the size of a half dollar was first noted in the left scapular area. This gradually enlarged and there was some discomfort when the patient lay on this area. Three or four months prior to admission, the patient thought that the enlargement was progressing more rapidly than before, he began to have a sense of ''pressure'' in the left side of the thorax, and the mass became slightly tender. He had lost no time from work, however. There had been a loss of 12 pounds (about 5.4 kg.) in the past year. Needle aspiration of the tumor had been carried out elsewhere, one week before the patient was seen at the clinic, with temporary reduction in the size of the tumor. The contents were not described. The results of a review of the history by systems were entirely negative.

On physical examination, the patient was found to weigh 145 pounds (about 65.8 kg.) and did not appear to be ill. The pertinent findings were as follows: A soft pulsatile mass, approximately 12 by 6 centimeters in size, was found extending from the inferior tip of the left scapula inferolaterally, with no discoloration of the overlying skin. This mass was not tender, and no thrill was palpable. On auscultation over this mass, a bruit with systolic and diastolic components was heard in some areas. It was best heard in the inferior lateral aspect of the mass. Pressure with the stethoscope over this area did not alter the character of the bruit. Blood pressure was 120/74. The pulse was 76 beats per minute.

The laboratory findings were: hemoglobin 13.2 Gm. per 100 c.c. (88 per cent); leucocytes 6,200 per cubic millimeter; serologic reaction for syphilis negative; sedimentation rate (Westergren) 9 mm. in 1 hour; total proteins 7.7 Gm. per 100 c.c.; normal albumin-globulin ratio; calcium 10.6 mg. per 100 c.c. of serum; phosphate 3.0 mg. of phosphorus per 100 c.c. of serum; Bodansky alkaline phosphatase was 2.8 units. The urine was negative except for rare erythrocytes in the sediment.

A roentgenogram of the thorax showed a soft-tissue tumor in the left side of the back, with scalloping defects involving the inferior margin of the seventh rib, both margins of the eighth rib, and the superior margin of the ninth rib (Fig. 1).

The clinical diagnosis was arteriovenous fistula and aneurysm of the posterior thoracic wall. Resection was advised in spite of the probable difficulty and danger involved, because it was felt that there was no alternative to surgical treatment. Accordingly, operation was carried out on July 14, 1950, by one of us (Clagett).

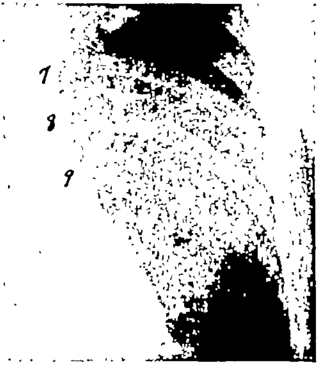

Fig. 1.—Portion of the preoperative roentgenogram of the thorax, showing the diffuse haziness of the soft-tissue mass in the posterior wall of the left side of the thorax, with scalloping defects involving the inferior margin of the seventh rib, both margins of the eighth rib, and the superior margin of the ninth rib.

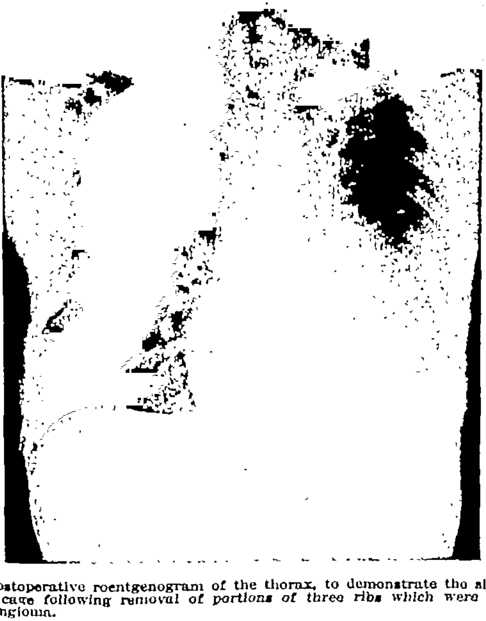

Fig. 2.—Postoperative roentgenogram of the thorax, to demonstrate the size of the defect in the thoracic cage following removal of portions of three ribs which were involved in the cavernous hemangioma.

Operation.—A curved incision was made over the large pulsating mass involving the left side of the thorax posteriorly in the region of the seventh, eighth, and ninth ribs, just below the tip of the scapula. The incision was deepened through the trapezius and latissimus dorsi muscles. The superficial layers showed some evidence of increased blood supply. The large pulsating tumor mass was exposed. It apparently involved the seventh, eighth, and ninth ribs and intercostal spaces and thoracic wall posteriorly. In order to ascertain the intrathoracic involvement, the ninth rib was sectioned anterior to the mass, and the pleura was opened. Huge tortuous dilated blood vessels involving a plaque of thoracic wall could be felt. The eighth and seventh ribs were sectioned anterior to the aneurysmal mass to find and ligate the intercostal vessels accompanying these three ribs, and that portion of the thoracic wall was then resected upward. The interior of the pleural space could then be exposed. With considerable difficulty, the seventh, eighth, and ninth intercostal arteries and veins adjacent to the aorta were dissected out, ligated, and divided. The whole tumor mass became softer after this, and resection of the plaque of involved thoracic wall was begun. In spite of the fact that it was thought that the entire blood supply to the thoracic wall had been controlled, on all sides, there was a very severe hemorrhage posteriorly as the aneurysmal mass was cut away. It came directly from the bone marrow of the three ribs where they had been cut posteriorly. This had to be controlled with bone wax. The whole area oozed and bled, but the hemorrhage was finally controlled with mattress sutures and gelfoam. The pleural space was washed with saline solution, and the lung was inflated. A catheter was inserted for temporary closed drainage of the pleural space. Since the defect in the thoracic wall was too large to permit closure, no attempt was made to close the opening between the ribs; however, the trapezius and latissimus dorsi muscles superficially were closed satisfactorily. An

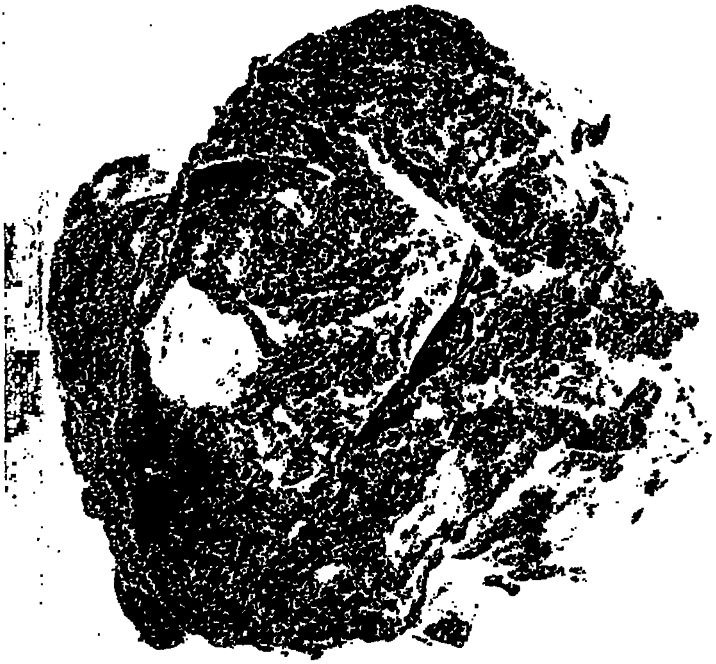

Fig. 3.—Gross pathologic specimen, consisting of a portion of the thoracic wall, showing the musculature overlying the portions of the resected ribs. Both the cavernous hemangiomatous musculature and ribs are involved in the mass.

Arteriovenous fistulas involving intercostal vessels are rare. Not a single case could be found among 447 cases of arteriovenous fistula reported in Callander's[17] series, nor among the 620 cases, the data on which were analyzed by Matas[18] from his own personal experience.

Notching or scalloping of ribs is usually associated with coarctation of the aorta. However, Batchelder and Williams[19] have described notching of the inferior surfaces of the fifth, sixth, and seventh ribs on the left, posteriorly, in the case of a man aged 38 years with the tetralogy of Fallot, in which at necropsy, dilated, tortuous intercostal arteries were found in these locations, without coarctation of the aorta or aneurysm of these vessels. Maier and Stout[12] also mentioned notching of the ribs, in a case of arteriovenous fistula of the thoracic wall in association with a pulmonary arteriovenous fistula, to which we have already referred. Maier mentioned this observation again, in discussing the report of Lindskog and his co-workers,[20] on pulmonary arteriovenous aneurysms. Our case illustrates this unusual cause for scalloping of the ribs yet again.

SUMMARY

A case has been reported of cavernous and capillary hemangioma of the left thoracic wall, with clinical evidence of arteriovenous fistulas, in which resection of the involved ribs, intercostal muscle bundles, and adjacent musculature of the thoracic wall was successfully carried out. The pertinent available literature is reviewed, and the significance of extracardiac bruits and of notching of the ribs, both of which were present in this case, is discussed.

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AN ANOMALY OF THE PULMONARY VEINS

A Case Study

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CONGENITAL malformations within the pulmonary circulation form a very interesting group of vascular anomalies. Once thought to be extremely rare and diagnosed only at autopsy, these lesions are being encountered more and more frequently by both clinician and surgeon. The increased use of exploratory thoracotomy, angiocardiography, and cardiac catheterization as diagnostic aids will no doubt bring many more of these cases to our attention. In the case reported here the right inferior pulmonary vein drained into the inferior vena cava. There were associated malformations of the lobar bronchi and an aberrant bronchial artery to the right lower lobe. The anomalies were discovered at operation and further evaluated by angiocardiography, cardiac catheterization, and bronchospirometry.

A 20-year-old, white male soldier was admitted to a station hospital in Japan because of a persistent shadow on roentgenogram examination which suggested segmental atelectasis in the right lower lobe. The patient had successfully undergone basic training and endured the rigorous physical exertions required of an infantryman without symptoms. He was transferred to Walter Reed Army Hospital for diagnosis and disposition. Roentgenograms revealed a crescent-shaped area of increased density in the right lower lung field, which paralleled the right heart border (Fig. 1).

Physical examination was negative. There was no history of cyanosis or dyspnea. The patient had developed normally in childhood, and his activities had in no way been curtailed. Pulmonary function studies were within normal limits. Bronchoscopy revealed some variation in the right lobar bronchi in that the lower lobe bronchus divided into two equal sized bronchi from which the segmental bronchi arose. One of the segmental bronchi seemed stenosed or occluded. The remainder of the bronchoscopic examination was negative. Bronchography substantiated the bronchoscopic findings (Fig. 2).

The more lateral of the two lower lobe bronchi gave off segments which seemed to correspond to the superior segmental bronchus of the lower lobe, the two segmental bronchi of the middle lobe, and the anterior basal and lateral basal bronchi of the lower lobe. The medial of the two main lower lobe bronchi gave off two segments which corresponded to the posterior basal and medial basal segmental bronchi. In addition, there were two segmental bronchi which appeared to be blocked or stenosed. It was on the basis of this evidence that the diagnosis of segmental atelectasis was made, and exploratory thoracotomy advised.

At operation, a few filmy adhesions were present between the lower lobe and diaphragm. As these were stripped away a large vein was visualized on the diaphragmatic surface of the lower lobe. The vein appeared to take origin from within the lung structure and pass through the central tendon of the diaphragm (Fig. 3).

Accompanying the vein was a large artery which entered the under surface of the lobe from below the diaphragm. Further exploration revealed complete absence of the pulmonary ligament and no inferior pulmonary vein in the region where it is ordinarily located. The superior pulmonary vein was easily visualized, and appeared normal. The pulmonary artery also appeared normal. In the region where bronchography led us to sus-

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pect stenosed or occluded bronchial segments, blind pouches from otherwise normal bronchi could be palpated. The anomalous vein which seemed to provide the only drainage for the right lower lobe was apparently the inferior pulmonary vein. As the exposed anomalous vein, which was approximately three centimeters in length, approached the diaphragm it was directly in line to join the inferior vena cava, which could be easily visualized because of the absence of the pulmonary ligament. The two veins united just beneath the diaphragm. Exploration of the right hemithorax revealed no other abnormalities. The heart appeared normal except for prominence of the right border. On the basis of these findings, it was decided that further studies which should include angiocardiograms and cardiac catheterization would be necessary to determine whether or not other abnormalities existed. The chest was closed. The patient had an uneventful convalescence.

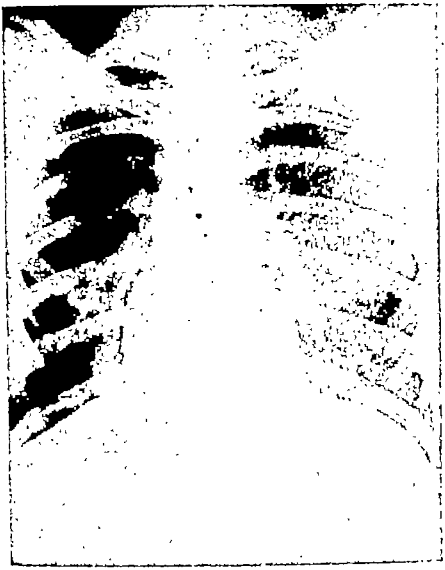


Fig. 1.—Posterior-anterior roentgenogram of the chest.

Angiocardiograms were done six weks after the exploratory thoracotomy. A series of eight angiocardiograms was made at one and one-half second intervals following diodrast injection (Figs. 4A and 5A). There were no abnormalities of the cardiac chambers, pulmonary arteries, or aorta. The left auricle was well visualized, and at the time of opacification of this chamber and the pulmonary veins, an unusual, irregular prominent network of vessels appeared in the right middle lung field continuous with a large vessel along the right cardiac border. The latter was approximately 18 millimeters in diameter and paralleled the curve of the right cardiac border from which it was separated by seven millimeters. This vessel seemed to pass beneath the diaphragm.

The filling of this vessel at this particular time leaves no doubt that it was a large pulmonary vein connected with an abnormally prominent and irregular network of pulmonary veins in the right middle lung field. No arteriovenous fistulae were demonstrated.

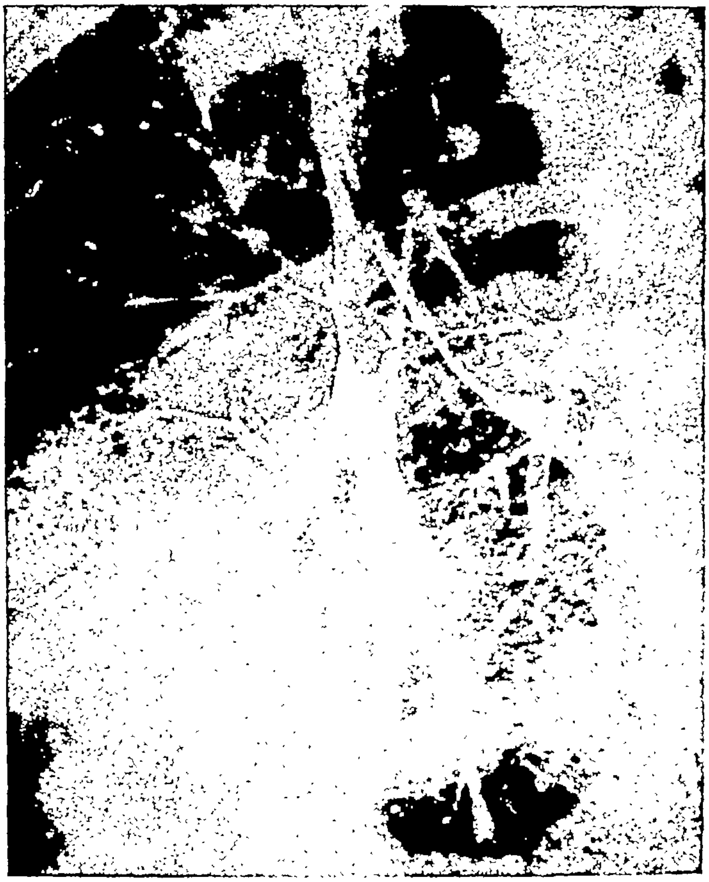


Fig. 2.—Bronchography—left anterior oblique.

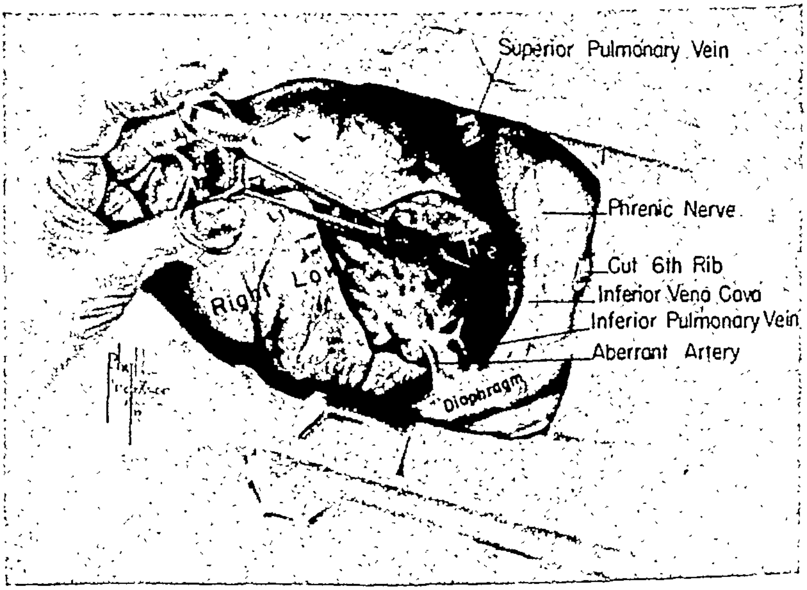
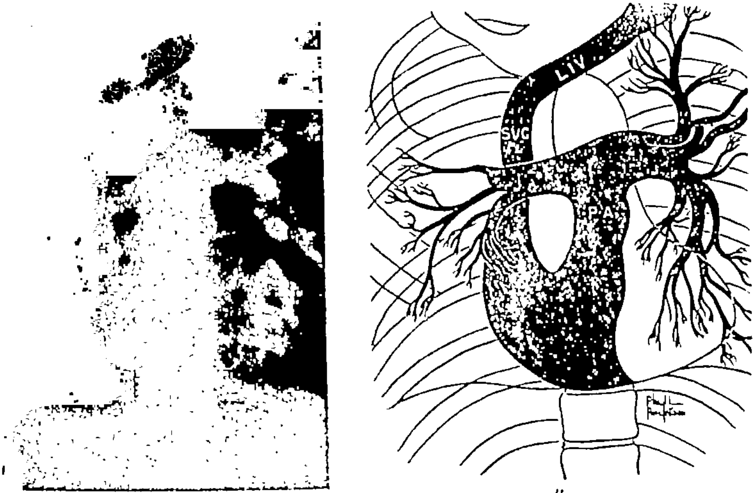
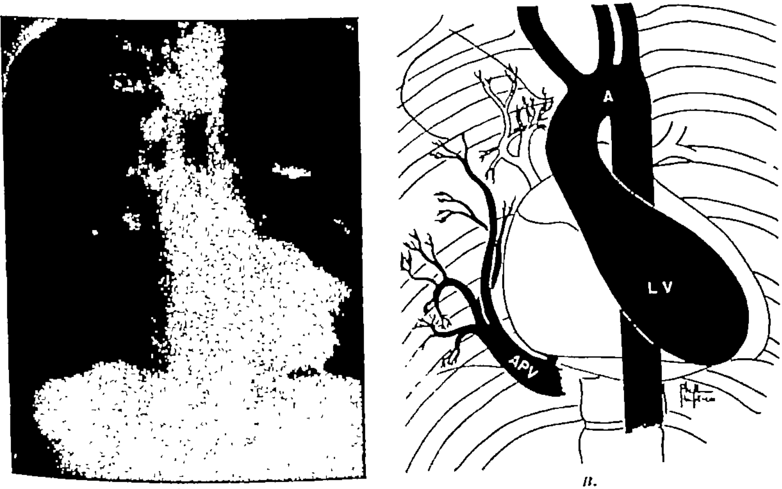


Fig. 3.—Artist's conception of the anomaly as seen at operation.



A. B.

Fig. 4.—Angiocardiogram three seconds after intravenous injection of 70 per cent diodrast showing opacification of superior vena cava, the right side of the heart, and the pulmonary arteries. *LIV*, left innominate vein. *PA*, main stem of pulmonary artery. *SVC* superior vena cava.



A. B.

Fig. 5.—Angiocardiogram seven and one-half seconds after intravenous injection of seventy per cent diodrast. The anomalous pulmonary vein becomes opacified at the same time as the left side of the heart and aorta. *A*, aorta. *APV*, anomalous pulmonary vein. *LV*, left ventricle.

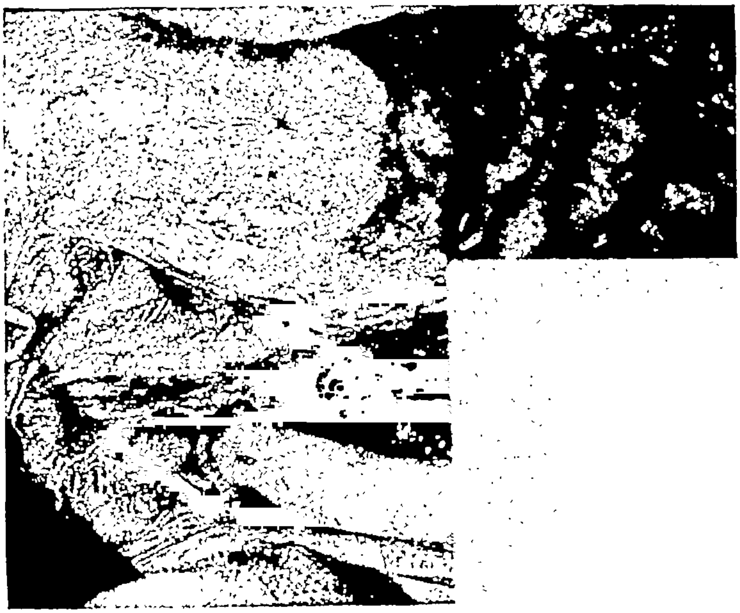


Fig. 5.—Photograph of gr

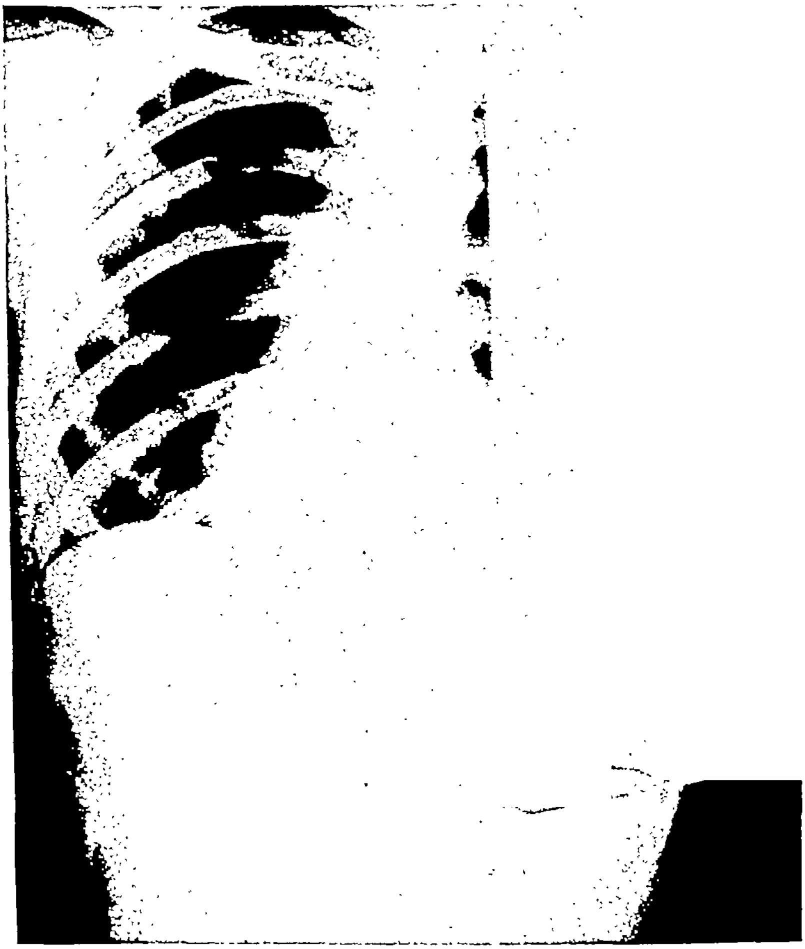


Fig. 6.—Posteroanterior roentgenogram taken on the ninth postoperative da

40-year-old man who had died from trauma twenty-three days previously. The graft had been preserved in a modified Tyrode's solution and kept at 4° C. A piece of the iliac artery which measured seven centimeters in length and fourteen millimeters in external diameter in its completely relaxed state appeared adequate for our needs. The coarctation (Fig. 4) was excised between Potts clamps, and the graft sutured in place, using continuous 0000 silk everting sutures. The distal end of the excised segment had been cut obliquely in order to preserve all branches of the abdominal aorta. As soon as the clamps were released, there *were excellent pulsations in the graft and the aorta below the graft.* Pulsations were palpable in the dorsalis pedis and the posterior tibial arteries. Fig. 5 illustrates the graft in situ.

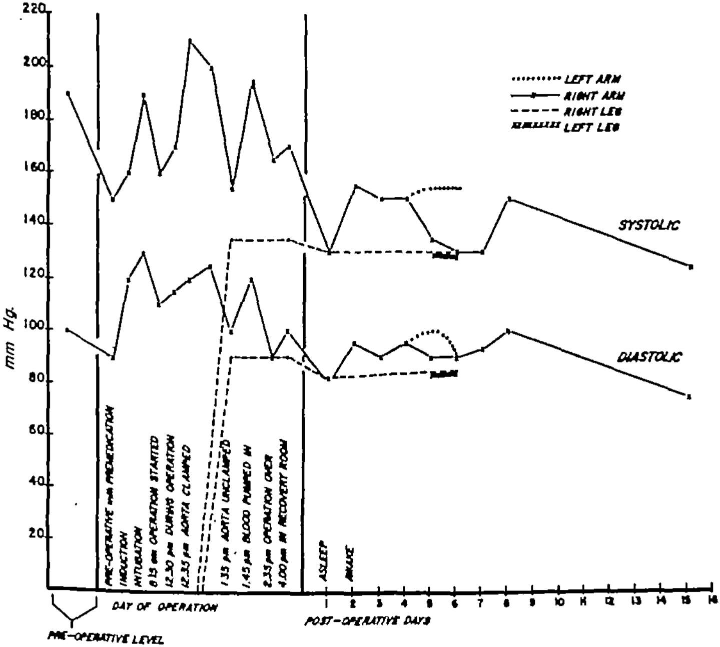


Fig. 7.—This chart depicts the changes in the patient's blood pressure.

The patient withstood the procedure well. Fifteen hundred cubic centimeters of blood were given during the operation.

Follow-up communication from the patient indicated that the blood pressure in her upper extremities has been maintained in the vicinity of 148/78. In the lower extremities the blood pressure has been approximately 123/88. The patient remarked that in general she has been feeling better than at any time for several years.

SUMMARY

This is the eighth case to be reported of coarctation of the thoracic aorta occurring at the level of the diaphragm, and the first case treated successfully with a preserved arterial graft.

The clinical features of hypertension in the arms with no palpable pulsations nor measurable blood pressure in the lower extremities combined with a systolic bruit of maximal intensity at the ninth and tenth thoracic vertebrae, fluoroscopic demonstration of a normal aorta as far as the middescending portion, and notching of the lower most ribs, suggested the diagnosis. Angiocardiography successfully demonstrated the correct location of the coarctation.

Thoracic exploration was postponed until preserved arterial grafts were available since the probability of a long stenosed segment was recognized. Adequate exposure was obtained by subperiosteal resection of the ninth rib. At operation a coarctation of the aorta six and one-half centimeters long was found which extended from the ninth thoracic vertebra to the first portion of the abdominal aorta. The stenosis was dissected free, excised, and replaced by a 23-day-old graft seven centimeters in length. A piece of the blood vessel used as a graft grew in tissue culture when it was thirty-nine days old. Postoperatively the patient convalesced uneventfully, and at the present time has relatively normal blood pressures in her extremities. (Figs. 6 and 7.)

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AN EFFECTIVE METHOD FOR BRONCHIAL CLOSURE

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ALTHOUGH bronchial closure following pulmonary resection has been fairly well standardized and the incidence of reopening has been greatly diminished, this unfortunate complication still occasionally occurs. A simple modification of the accepted methods of covering the stump with pleura has proved to be so successful in the past year that it is thought to be worth while briefly to present the technique used.

The experience with this technique of bronchial closure is too limited to be of statistical significance, but from a practical standpoint it seems well worth reporting. It has been used in thirty-five cases of pulmonary resections. These were lobectomy, segmental resection, and pneumonectomy for a variety of conditions including neoplasms and infections, both nontuberculous and tuberculous. There was no evidence of bronchial disruption in any case, but in one there was evidence of leak. In the latter a leak was assumed because of the development of an empyema with some production of bloody mucus several weeks following a left lower lobectomy and lingulectomy for bronchiectasis. The empyema was drained openly and satisfactory healing occurred without evidence of persistence of a fistula.

Rienhoff and associates[1] in 1942 reviewed the subject of bronchial closure and drew important conclusions based on experimental and clinical observations. They stressed the important factors concerned with the healing of the severed bronchus. These were that the cut bronchus heals at its end, the importance of preserving a viable stump by avoiding trauma to it or to any of its vessels, and the importance of covering the cut end of the bronchus with adjacent mediastinal pleura. They obtained healing by several methods: one of which was simply to leave the bronchus widely open with pleura sutured against it; another was to place the suture line as far proximal as possible from the cut end, covering the latter with pleura only. Their most frequent and serious complication was opening of the bronchial stump with the development of a fistula.

In 1945 Sweet[2] presented a method of bronchial closure based on the above principles and stressed, among other things, the importance of covering the cut end of the bronchus with pleura by suturing the edge of a pleural flap to the peribronchial tissue proximal to the sutured end.

A method of bronchial closure which, although it may not be original, has proved entirely satisfactory during the past year, is presented.

The method used is as follows: the part to be resected is first mobilized down to its bronchus by dissection and division of its vascular supply and peri-

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bronchial attachments, except that in segmental resection the vessels and bronchus may be isolated first. The bronchus is then clamped close to the lung tissue distal to the point of planned section to prevent any spillage from the diseased portion. Sutures of No. 0000 silk on small straight needles are then passed through the bronchus about 3 mm. proximal to the line of section. This entire line of marginal sutures is placed, keeping each suture in order. Gauze is then placed around the bronchus to guard against any possible contamination.

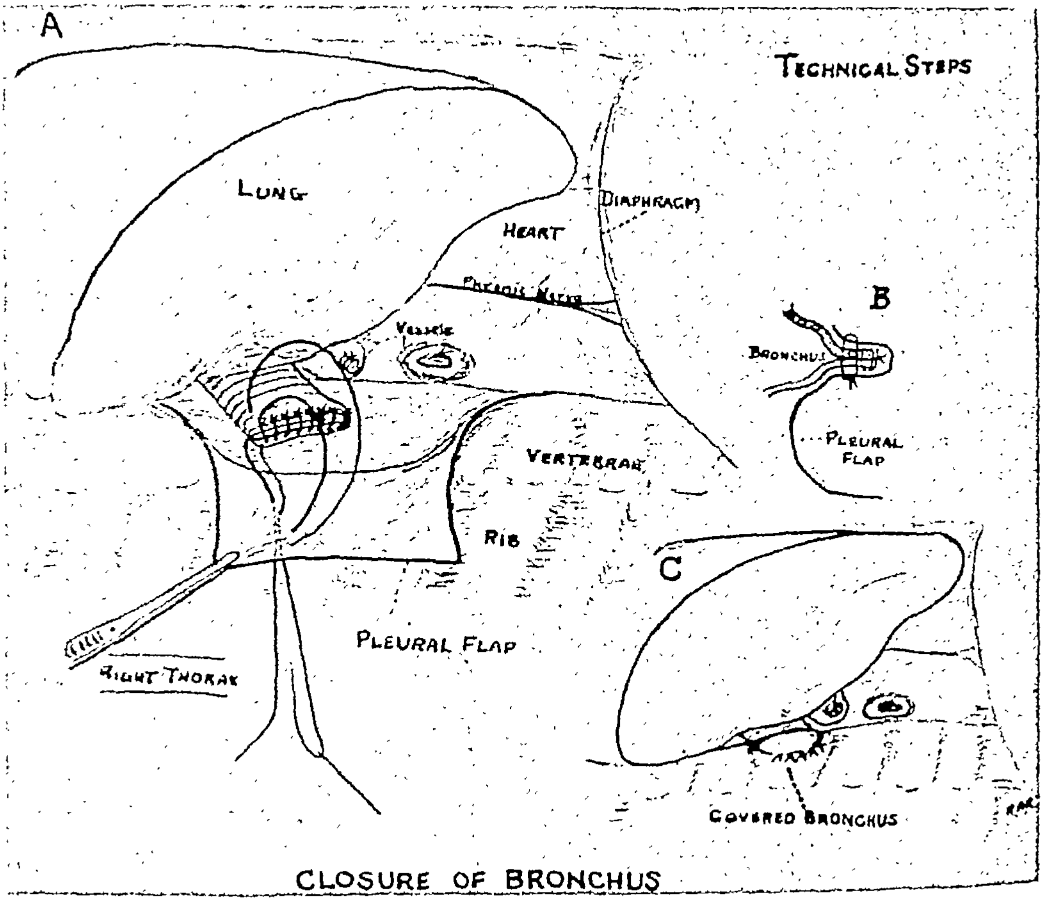

Fig. 1.—Steps in closure. *A*, First vertical mattress suture in place proximal to marginal row of bronchial sutures; others to follow in order. *B*, Section of bronchus showing sutures in place and tied. *C*, Completed closure.

These sutures are then held aside while the bronchus is cut across directly with the knife, or with a right angle scissors, and the sutures are then held up snugly as a unit. The bronchus is thus open a very short time. The sutures are then tied snugly, one by one, approximating the membranous portion to the cartilaginous portion of the bronchus. One or two additional sutures may then be placed if necessary for an airtight closure. A variation of this part of the procedure is to place and tie each suture as the bronchus is cut across as described by Sweet (1945). The straight needle is believed to be better than a curved because the point is more easily and definitely directed.

The marginal row of sutures is used to manipulate and to hold the bronchus while it is covered with adjacent pleura, this being elevated as a flap, or simply shifted, usually from the posterior vertebral area. In Fig. 1, *A* several vertical mattress sutures of fine silk are placed through the bronchus just proximal to the marginal row of sutures, each of these sutures anchoring the pleura snugly over the bronchus both anteriorly and posteriorly in glovelike fashion. The nonserous surface of the pleura is brought closely against the cut end of the bronchus. Each suture is passed through an edge of the pleura, then through the bronchus, and through the other edge of the pleural flap. It is then brought directly back alongside itself through pleura, bronchus, and pleura close to the starting point. The pleura is brought nicely over the entire cut end of the bronchus as these sutures are tied after all have been placed (Fig. 1, *B* and *C*).

This method of closure approximates the bronchial mucosa proximal to the cut end of the bronchus and the marginal sutures. Though this may be a disadvantage, it has not thus far proved to be so. The method does tend to bring the subpleural areolar tissue in close apposition to the end of the bronchus without leaving appreciable, actual, or potential space between it and the pleura. This should further insure good healing.

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A BRONCHIAL YARDSTICK

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WITH the development of thoracic surgery, in recent years, a need for an easy and accurate means of obtaining linear measurements within the tracheobronchial tree has been increasingly apparent. For an example, one of the things a chest surgeon needs to know is how much distance there is between a bronchial lesion and the carina. For the purpose of surgery a report from the endoscopist to the effect that it is so many centimeters from the incisor teeth, or an estimate of two to three centimeters beyond the carina is of little value. The thoracic surgeon has been justified in his criticism of such reporting, and it has forced him, in many instances, into doing his own bronchoscopy when he is already carrying a too heavy work load.

With the hope of starting endobronchial studies on a more satisfactory basis from the point of view of accurate quantitative examination the Bronchial Yardstick has been designed. It allows the endoscopist quickly, easily, and safely to obtain the necessary measurements without undo prolongation of the diagnostic bronchoscopy.

The instrument consists of a brass rod (or tube) 1.5 mm. in diameter and 60 cm. in length. On this rod are mounted, in unattached tandem, two jewelers' pin clutches of diameter suitable to the lumen of the bronchoscope to be used, so vision by them is possible. These pin clutches are mounted in direction such that they are locked in the direction of the peripheral end of the rod, and yet will move easily up the rod, thus eliminating danger of their dropping off the end of the rod into the tracheobronchial tree.

The proximal pin clutch is fitted with a transverse spike so that as the instrument is carried into the bronchoscope the clutch is automatically carried up the rod and away from the peripheral clutch. A small fitting at the end of the rod serves two purposes: (1) to prevent possible escape of a faulty pin clutch, and (2) it acts as a sort of gun site making it easier to accurately place the end of the rod at the desired spot, i.e., the border of the lesion in question.

The technique of bronchial linear measurements with the above described instrument is easy, and to the experienced endoscopist the diagramatic illustration (Fig. 2) will be almost self-explanatory. The two pin clutches are set at the point desired, the rod is then advanced into the trachea or bronchus to the further extremity of the line between the two points in question. The instrument is then withdrawn and the distance between the two pin clutches is measured in millimeters. Thus, it is possible, by successive settings of the clutches, not only to measure the distance from the carina to the lesion, but also the size of the lesion, in respect to the length of the bronchus, may also be measured.

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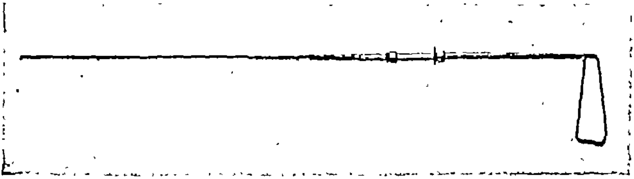

Fig. 1.

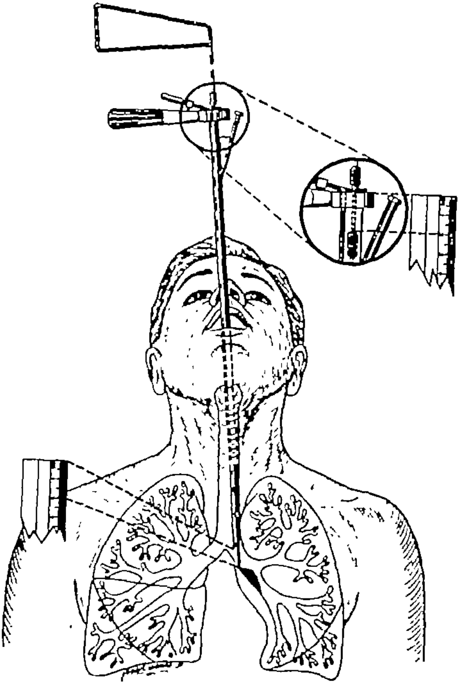

Fig. 2.

SUMMARY

1. An instrument, termed a Bronchial Yardstick,* for obtaining linear measurements within the tracheobronchial tree has been described.
2. The instrument has been found satisfactory as to ease and safety in actual use.
3. The accuracy of the measurements obtained are satisfactory to the thoracic surgeons.
4. It is hoped that the introduction of this instrument may mark the beginning of more accurate endoscopic reporting.

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*The Bronchial Yardstick may be obtained at The Foregger Company, Inc., 55 West 42nd Street, New York 18, N. Y.

CLINICALLY INDETERMINATE PULMONARY FIBROSIS
A PATHOLOGIC STUDY*

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IN THE study of pulmonary disease the physician is often confronted with the problem of diagnosis and treatment of a patient who has shortness of breath, cough, and at times cyanosis, either with or without positive physical signs. The roentgenogram of the thorax shows bilateral parenchymal infiltration of indeterminate nature, and additional study may fail to reveal the underlying cause. The term "pulmonary fibrosis" is often used to categorize these patients.

Mallory[1] defined pulmonary fibrosis as "A condition characterized by multifocal or generalized fairly evenly disseminated pulmonary lesions, the origin of which is dubious." Such a definition is supported clinically by the roentgenogram of the thorax, which presents the only uniformly objective evidence of pulmonary disease.

It is only by adequate pathologic study that any type of classification can be made. Mallory,[1] in a review of the records of 6,000 necropsies, found the anatomic diagnosis of pulmonary fibrosis in fifty-nine instances, in only twenty-three of which the patient had had any pulmonary symptoms. He divided these cases into four groups, as follows: (1) organization of intra-alveolar fibrinous exudate, as seen in bronchitis or pneumonic exudation without resolution; (2) healed infarction; (3) healed destructive pneumonitis and infections producing necrosis, as seen in infectious granulomas, and septic mixed infections which are usually associated with abscess formation or bronchiectasis; and (4) interstitial fibrosis, either in alveolar walls or lymphatic channels. In this group are mentioned prolonged chronic passive congestion from valvular cardiac disease, interstitial pneumonia, sarcoid, viral or rickettsial infections, chemical irritation, as well as still other processes.

Some of the characteristics of the lung, said to have fibrosis, and regardless of the specific etiologic agent, are increase in the collagenous tissue, and destruction and distortion of the elastic elements with loss of the normal recoil power of the parenchyma. The alveoli are obliterated which in turn decreases the area of ventilating surface. The scarred walls have decreased vascularity in the end stages, which produces diminished aeration of blood. Metaplasia of the alveolar lining cell to a cuboidal type may occur, leading to an alteration in function of the alveolar lining.

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In 1932, Cooper[2] tabulated twelve conditions which must be considered in the differential diagnosis of bilateral, fairly evenly disseminated lesions. Austrian and Brown,[3] in 1942, listed twenty-five causes for such a roentgenographic picture, and by 1948, Nelson and Heublein[4] mentioned seventy-five different causes. An etiologic classification of pulmonary fibrosis, as modified from a summary by King,[5] is presented in Table I.

In an attempt to clarify the diagnostic term "pulmonary fibrosis," we made a study of cases in which a clinical diagnosis of bilateral diffuse pulmonary fibrosis of indeterminate origin had been made and in which necropsy specimens were available. Details of the study follow.

SELECTION OF CASES

A review was made of the cases at the Mayo Clinic from Jan. 1, 1920, through Feb. 28, 1950, in which a clinical diagnosis of bilateral pulmonary fibrosis or indeterminate bilateral pulmonary infiltration was made, and in which the roentgenograms of the thorax revealed disseminated lesions. All cases in which the etiologic agent was known or strongly suspected were excluded from this series, as were those in which the data was insufficient for adequate study. No cases in which an occupational hazard might have contributed to the "fibrosis" were included. Diseases such as Boeck's sarcoid, asthma, senile emphysema, proved or probable bronchiectasis, chronic bronchitis, tuberculosis, or other proved parenchymal infections were not included. Those cases of fibrosis associated with rare systemic diseases such as scleroderma or xanthomatosis were excluded. The only other criterion for selection was whether or not necropsy had been performed. Most of the instances of indeterminate diagnosis could not be included in the detailed study, because of lack of postmortem examination, or because necropsy had been done elsewhere and the findings were not known to us. An attempt was made to select only those cases in which the clinician had been unable to make or suggest a definite diagnosis.

TABLE I. ETIOLOGIC CLASSIFICATION OF PULMONARY FIBROSIS

Infectious diseases	Circulatory disturbances
Inhalation diseases	Skeletal abnormalities
Neoplastic diseases	Postradiation changes
Diseases of blood	Emphysema, asthma, asthmatic bronchitis
Systemic diseases	Indeterminate
Bronchiectasis	Indeterminate

The cases records of 647 patients were reviewed. For fifteen of these patients there were adequate clinical data and necropsy material, and the cases are reported herein.

REPORT OF CASES

CASES 1, 2, and 3.—*Infectious Diseases:* Two patients had pathologic evidence of organized pneumonia. One was a 51-year-old woman post-office worker (Case 1), and the other a 41-year-old salesman (Case 2), both with symptoms of progressive dyspnea, and slight cough for four years and fourteen months, respectively. The roentgenograms of the chest are shown in Figs. 1*a* and 1*c*, respectively.

Microscopically, in each case there were marked replacement of alveolar exudate by fibrous tissue and obliteration of alveolar spaces (Fig. 1*b*). Alveolar and bronchial epithelial hyperplasia, alveolar hyaline membrane (Fig. 1*d*), and generalized, moderately severe vascular change consisting of thickening of the wall of pulmonary arterioles, with reduction in the size of the lumen, were other findings.

These patients suffered from gradual replacement of the functioning pulmonary bed by scar tissue. There was no history of an acute infectious process in Case 1, and the disease was not febrile until the last few days of life. There had been no known exposure to

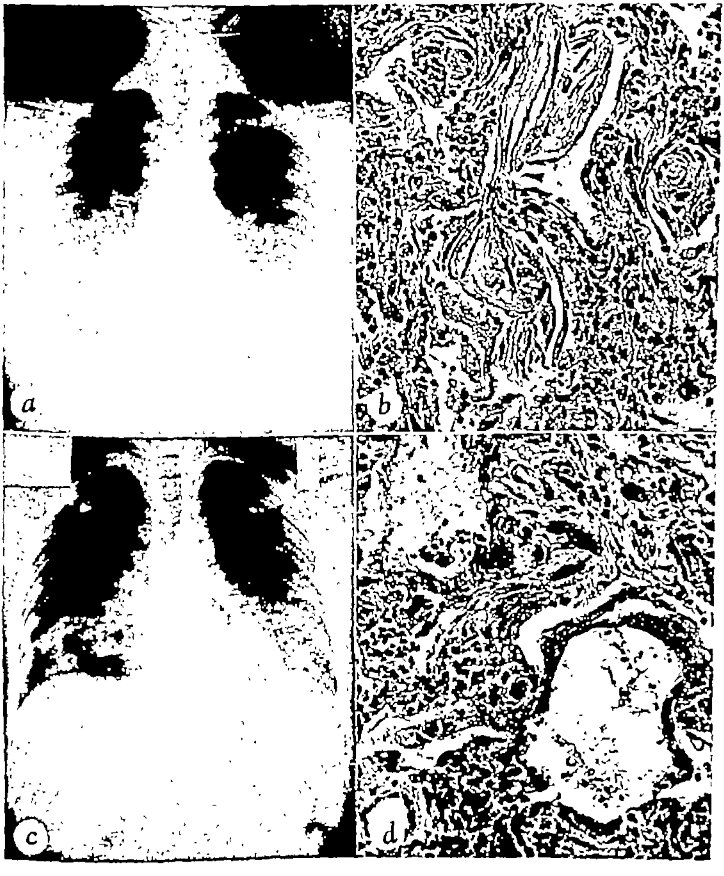

Fig. 1.—Organized pneumonia. *a* (Case 1). Roentgenographic appearance. *b*, *c*, and *d* (Case 2). *b*, Pores of Cohn and sheaves of Newman are shown (hematoxylin and eosin × 195). *c*, Roentgenographic appearance. *d*, Alveolar hyaline membrane (hematoxylin and eosin × 195).

irritants. No pathogenic organism was recovered during life, and at postmortem examination no bacteria were found in the lesions. The pathologic picture was one of an organized pneumonia. In the second case there was a history compatible with a bronchopneumonia at the onset of the illness, which is in contrast to Case 1.

The third case of infection was that of a 34-year-old salesman with a two-year history of cough, progressive dyspnea, weakness, and loss of weight (Case 3). Necropsy revealed extensive pulmonary scarring, calcification, and necrosis. All sections contained a fungus (Fig. 2). Cultures of the lungs yielded *Aspergillus glaucus.**

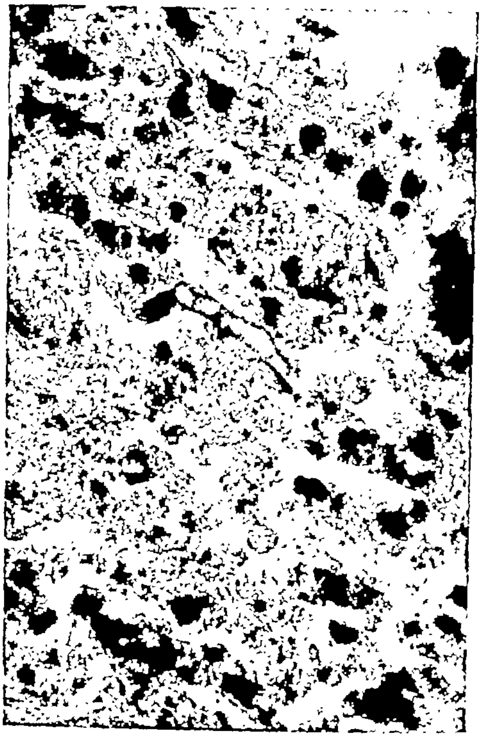

Fig. 2.—(Case 3). Fungus (Aspergillus glaucus) in pulmonary exudate (hematoxylin and eosin × 850).

CASES 4, 5, 6, and 7.—*Neoplastic Disease:* There were four cases in which neoplastic disease was found at necropsy. A 28-year-old man (Case 4) had respiratory symptoms for two months, and vague abdominal pain for one year. The roentgenogram of the chest is shown in Fig. 3*a*. He died of respiratory failure. A primary adenocarcinoma of the transverse colon was found at postmortem examination. Microscopic sections showed tumor cells distending lymphatic vessels throughout all pulmonary lobes (Fig. 3*b*). There was very little fibroblastic proliferation to explain the clinical impression of pulmonary fibrosis.

A 63-year-old farmer (Case 5) had respiratory complaints for three months (Fig. 3*c*) and vague abdominal distress for six months. The pathologic findings included carcinoma of the tail of the pancreas with metastasis to the lungs (Fig. 3*d*), liver, and right adrenal. In this case also there was no fibroblastic proliferation in the parenchyma.

A 43-year-old housewife (Case 6) had incapacitating dyspnea for nearly three months. The roentgenogram of the thorax (Fig. 4*a*) showed a bilateral indeterminate process. Postmortem examination revealed a primary carcinoma of the stomach with lymphatic metastasis to the lungs. There was marked replacement of parenchyma by metastatic carcinoma with fibrous stroma (Fig. 4*b*), in contrast to the previous cases.

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*We are indebted to Dr. J. L. Marshall of Aspinwall Pa., for the report of necropsy and the loan of microscopic sections.

A 33-year-old man (Case 7) complained of cough, shortness of breath, and fever for one year. Pathologic examination showed a lymphoma of the Hodgkin's type involving the lymph nodes, liver, lungs (Fig. 4*c*), spleen, adrenals, and bone marrow.

CASE 8.—*Disease of the Blood:* A 62-year-old farmer had symptoms for only five weeks. The peripheral blood picture was compatible with a diagnosis of aplastic anemia, while the roentgenogram of the thorax revealed a diffuse bilateral infiltration in the lungs (Fig. 5*a*).

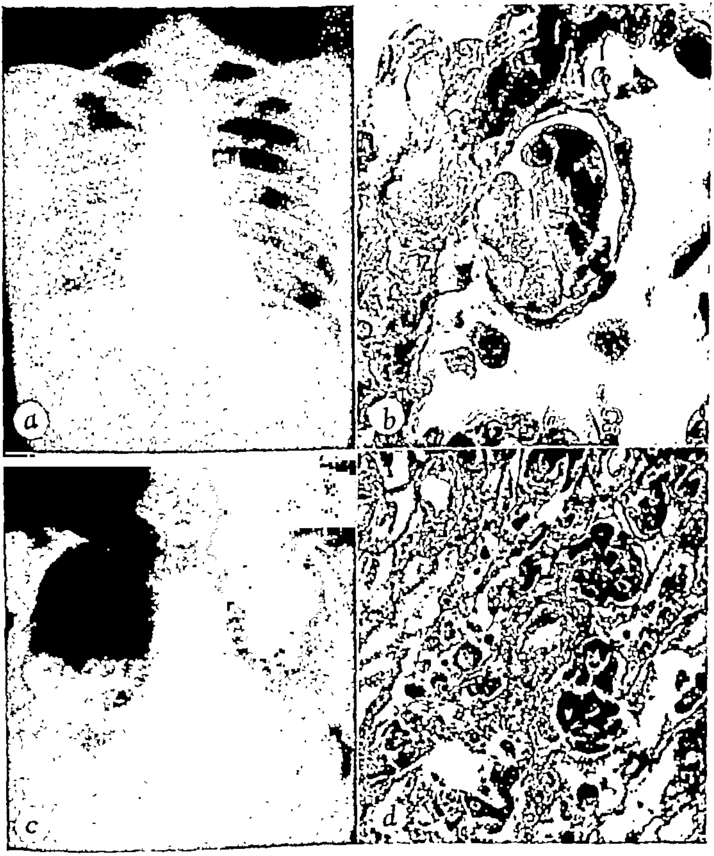

Fig. 3.—Metastatic malignant lesions in the lung. *a* and *b* (Case 4). *a*, Roentgenographic appearance. *b*, Metastatic carcinoma in a small lymphatic vessel of lung (hematoxylin and eosin × 760). *c* and *d* (Case 5). *c*, Roentgenographic appearance. *d*, Tumor cells in pulmonary alveoli (Mucin × 55).

Microscopically there were multiple focal areas of lymphocytes scattered in the alveolar walls throughout both lower lobes (Fig. 5*b*).

CASES 9, 10, 11, and 12.—*Bronchiectasis:* On pathologic examination in four cases there were extensive bronchiectasis, chronic bronchitis, patchy organized pneumonia, and varying degrees of emphysema and fibrosis (Figs. 5*d* and 6*b*). There were three women and one man, whose ages ranged from 54 to 68 years. Symptoms of cough, expectoration, and dyspnea

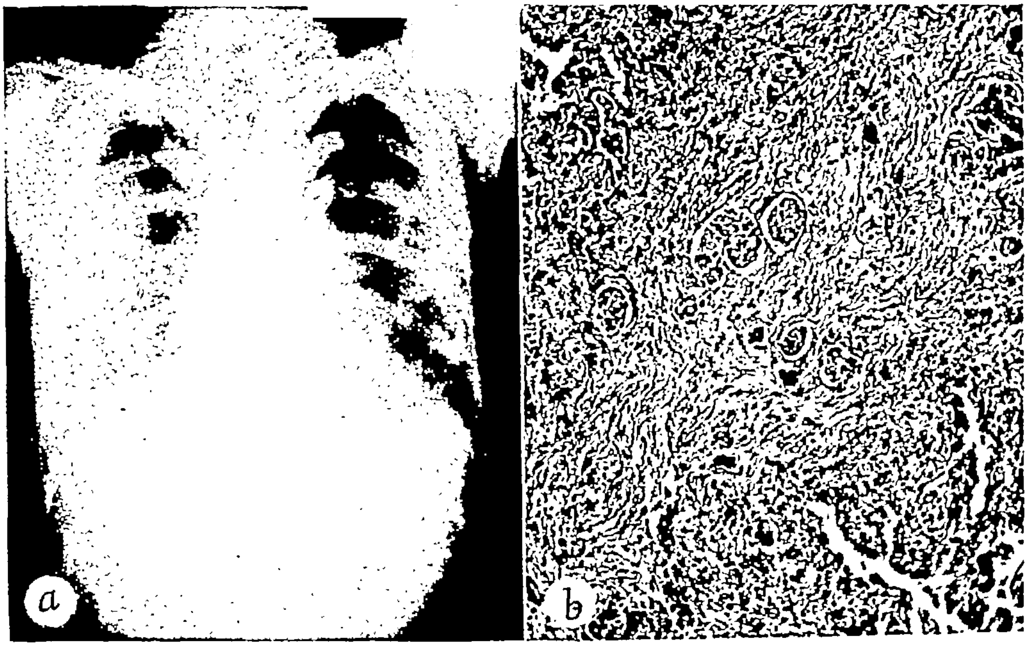

Fig. 4.—Metastatic malignant disease of the lung. *a* and *b* (Case 6). *a*, Roentgenographic appearance. *b*, Metastatic carcinoma with fibrous stroma (hematoxylin and eosin × 100).

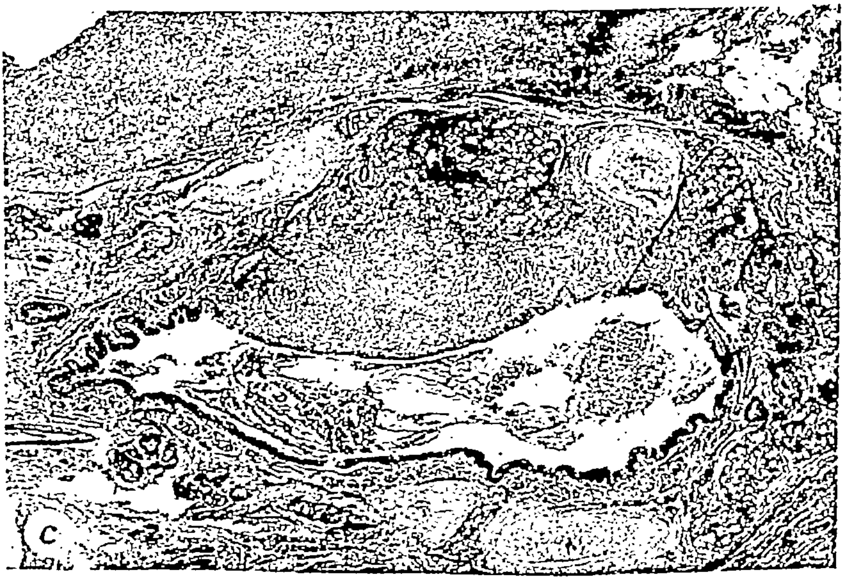

Fig. 4.—*c* (Case 7). Invasion of the bronchial wall by lymphoma of the Hodgkin's type (hematoxylin and eosin × 14).

had varied in duration from one to six years. Roentgenograms of the thorax are shown in figures 5*c*, 6*a*, and 6*c*. The most prominent symptom in this group of patients had been severe progressive dyspnea, even to the point of respiratory failure.

It is of interest that none of these patients gave a history characteristic of bronchiectasis, and all were in an older age group. It is presumed that the pathologic findings were on the basis of chronic low-grade infection producing severe destruction of pulmonary tissue and replacement fibrosis.

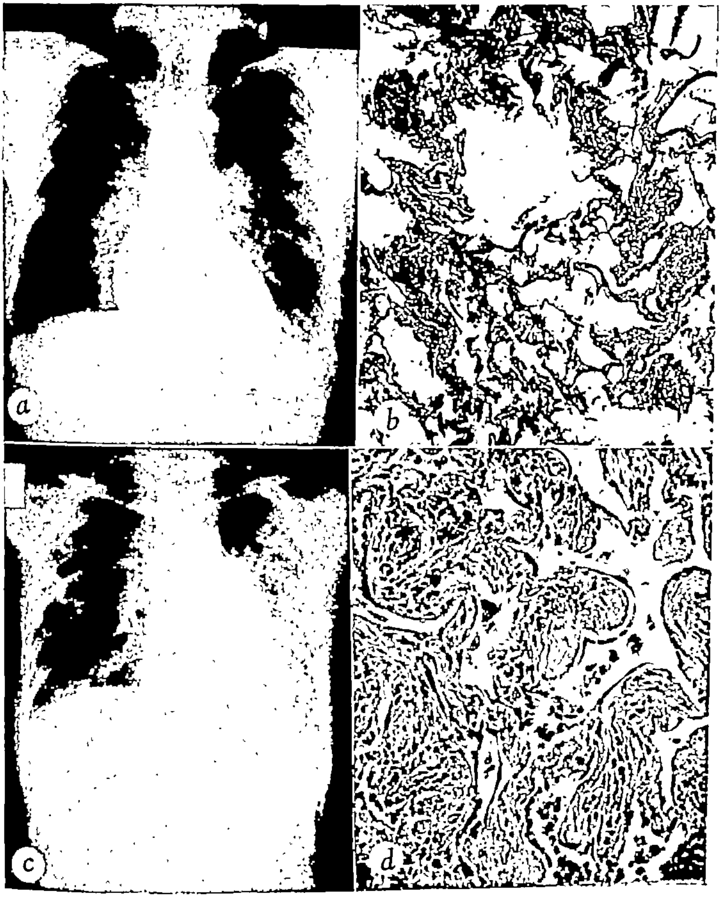

FIG. 5.—*a* and *b* (Case 8). *a*, Roentgenogram of the thorax. *b*, Focal areas of lymphocytes within the pulmonary parenchyma (hematoxylin and eosin × 43). *c* and *d* (Case 9). Bronchiectasis. *c*, Roentgenographic appearance. *d*, Bronchiectasis, parenchymal fibrosis, and cuboidal changes of the alveolar epithelium (hematoxylin and eosin × 130).

CASES 13 and 14.—*Circulatory Disturbance:* One patient in this group presented anatomic evidence of rheumatic heart disease with mitral stenosis (Case 13). This patient was a 24-year-old woman student who had symptoms of progressive dyspnea and cough for one year. The results of physical examination of the heart were negative except for tachycardia, and there were basal pulmonary râles. A roentgenogram of the thorax (Fig. 7*a*) showed a diffuse infiltration in both lower pulmonary fields and pleural effusion. Microscopically the lungs revealed a severe degree of chronic passive congestion with marked thickening of the alveolar walls, dilated capillaries, and increased number of fibroblasts and reticulum fibers

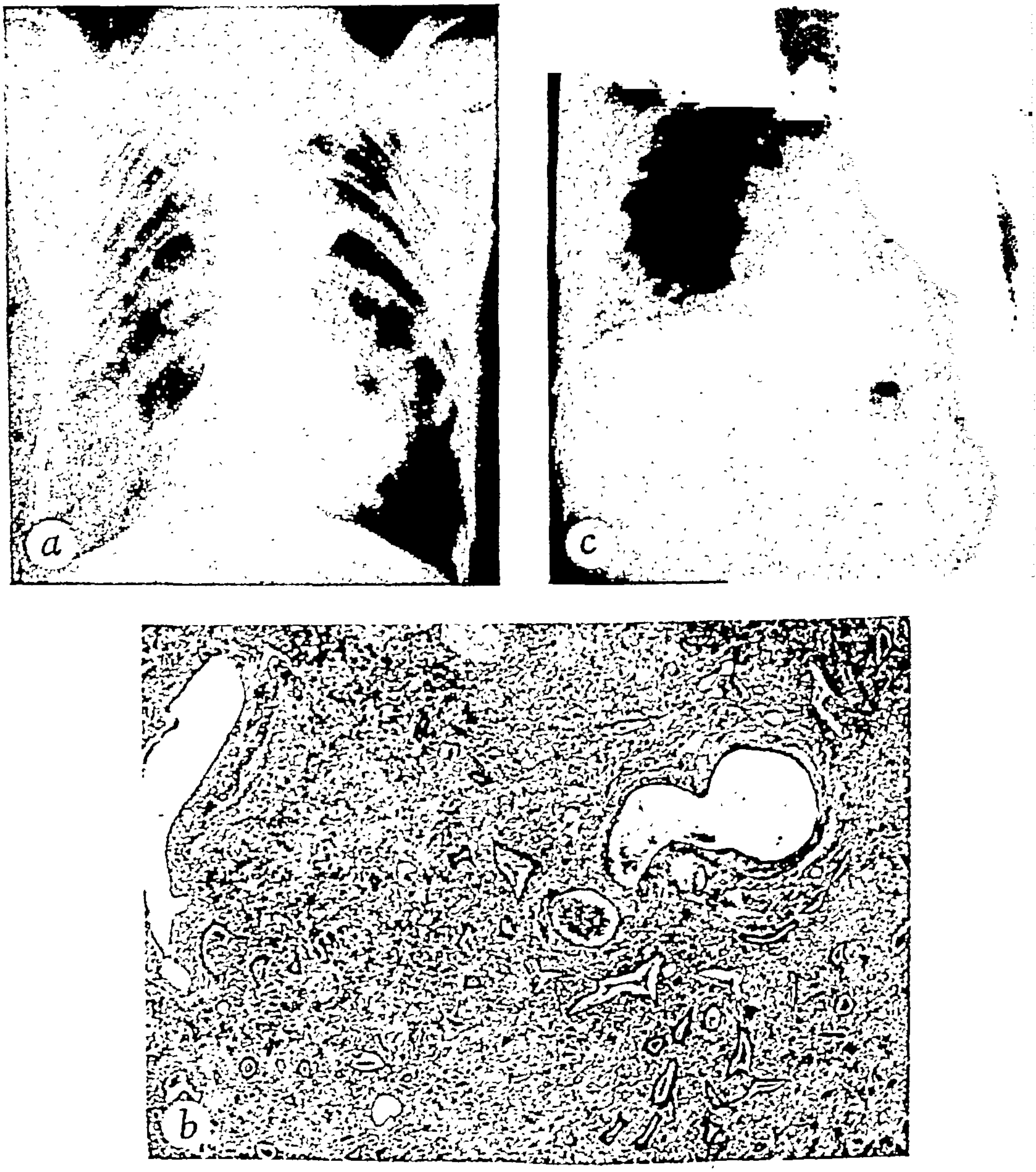

Fig. 6.—Bronchiectasis. *a* and *b* (Case 11). *a*, Roentgenographic appearance. *b*, Bronchiectasis and marked scarring of the pulmonary parenchyma (hematoxylin and eosin × 65). *c* (Case 12), Roentgenographic appearance.

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Original Communications

A CONSIDERATION OF SOME OF THE PROBLEMS IN CARDIOVASCULAR SURGERY

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During the last several decades there has been a progressive increase in deaths attributable to cardiovascular disease. It is estimated[1] that 44 per cent of all deaths in the United States in 1948 were due to disorders of the heart and vessels, and that there are at present nine million people in this country with some type of disease of the cardiovascular system. As the length of life increases, derangements of this system assume added importance. Hypertension, arteriosclerosis, and rheumatic fever make up the great bulk of cardiovascular problems, and, unfortunately, much remains to be learned about the etiology as well as the prevention and treatment of these diseases. It is to be hoped in the future that appropriate preventive measures will greatly reduce the mortality and morbidity of cardiovascular disease in general. Even if this hope is realized surgery will still have a role to play, particularly in the treatment of the complications of a mechanical nature.

Excluding peripheral vascular diseases such as arteriosclerosis, Raynaud's disease, Buerger's disease, and varicose veins, the major proportion of cardiovascular cases now amenable to surgery are of congenital or traumatic origin. Trauma, the cradle of surgery, will continue to contribute its quota, even if world conflicts should disappear. Preventive measures such as the possible elimination of rubella may result in a decrease in the incidence of congenital heart disease, but the chances are that congenital disorders will be of fairly frequent occurrence for centuries to come.

It would be unfair to the field of cardiovascular surgery to judge its future potentialities on the basis of present accomplishments, since this field is as much an advancing one as is that of preventive medicine and that of nonoperative therapy. For example, a fundamental advance which would solve the problem of homotransplantation of tissue would open up great potentialities in cardiovascular surgery.

This lecture is not intended to be an all-inclusive consideration of the subject. For example, peripheral vascular disease and portal hypertension will

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Presidential Address, Thirty-first Annual Meeting of The American Association for Thoracic Surgery, Atlantic City, April 16, 17 and 18, 1951.

not be discussed. Greater attention will be devoted to congenital heart disease because of the recent advances in this field. I have arbitrarily divided the disorders to be considered into three groups: (1) those in the treatment of which surgery may bring about good or excellent results, (2) those in which surgical treatment may cause moderate improvement, and (3) those in which surgical treatment is of doubtful value or is as yet undeveloped. There is a justifiable difference of opinion as to the group in which some of the disorders should be placed. The main emphasis will be on unsolved problems rather than on accomplished facts.

The conditions will be discussed in the following order:

I. Disorders in the Treatment of which Surgery may Accomplish Good or Excellent Results
  A. Acquired Lesions
    1. Constrictive Pericarditis
    2. Wounds of the Heart
    3. Systemic Arteriovenous Fistula
    4. Arterial Aneurysm
    5. Mitral Stenosis
  B. Congenital Lesions
    6. Patent Ductus Arteriosus
    7. Coarctation of the Aorta
    8. Pulmonary Stenosis and Atresia
    9. Pulmonary Arteriovenous Fistula
    10. Anomalies of the Aortic Arch
II. Disorders in the Treatment of which Surgery may Bring about Moderate Improvement
  A. Acquired Lesions
    11. Essential Hypertension
  B. Congenital Lesions
    12. Transposition of the Aorta and Pulmonary Artery
    13. Anomalies of Venous Return
III. Disorders in the Treatment of which Surgery is of Doubtful Value or Suitable Methods are not yet Fully Developed
  A. Acquired Lesions
    14. Coronary Arterial Disease
    15. Aortic Valvular Stenosis
    16. Insufficiency of Heart Valves
    17. Intracardiac Tumors
  B. Congenital Lesions
    18. Anomalous Origin of Left Coronary Artery
    19. Auricular Septal Defects.
    20. The Lutembacher Syndrome
    21. Ventricular Septal Defects
    22. The Eisenmenger Complex
    23. Pulmonary Hypertension

I. DISORDERS IN THE TREATMENT OF WHICH SURGERY MAY ACCOMPLISH GOOD OR EXCELLENT RESULTS

A. Acquired Lesions.—

1. Constrictive Pericarditis: This is now a well-recognized clinical entity which usually presents few difficulties in diagnosis. It is often impossible to determine the cause. In most such cases it is likely that the tubercle bacillus is the offender. The use of chemotherapeutic and antibiotic agents in the prevention and control of infections will probably result in a reduction in the incidence of constrictive pericarditis.

The fundamental surgical problem remains the same as that enunciated by Delorme,[2] namely, the removal of the scar which is constricting the heart and in some cases the great blood vessels. It is necessary to remove the scar overlying the epicardium as well as that which lines the pericardium.

Prior to the discovery of streptomycin and similar antibiotic agents, it was held that patients with tuberculous constrictive pericarditis should not be operated upon until the process became inactive. One of the reasons for this viewpoint was that operation subjected the patient to the dangers of a systemic as well as a localized spread of the disease. A second reason given was that it is extremely difficult to remove the shaggy exudate that is present in the subacute stage. It is the opinion of Holman[3] that antibiotic therapy may now allow one to operate with safety during the earlier stages of the disease. Certainly these agents will lessen the chances of spread of the disease, and possibly it will not be necessary to remove the tuberculous granulation tissue covering the surface of the heart. No one has yet had sufficient experience with the combined use of surgery and antibiotic therapy in the treatment of active tuberculous pericarditis to give an authoritative answer.

The types of incision that may be employed in decortication of the heart include a parasternal one with resection of costal cartilages, a median sternotomy, and an intercostal transpleural approach. At the present time I prefer the latter incision for it gives more adequate exposure than the others. A long left anterolateral incision is made overlying the fourth interspace, and the fourth and fifth costal cartilages are divided at their sternal attachments. Through this incision one can expose and decorticate both the left and the right side of the heart, including the superior and inferior vena cava. It makes little difference if the right pleura is torn, provided one recognizes the condition and maintains inflation of the lung. Recent experience has shown that thorough decortication usually causes a prompt decline in the venous pressure and general improvement of the patient. Our results with this incision are much better than those obtained when a parasternal extrapleural incision was used.

2. Wounds of the Heart: As has been true of developments in many fields of surgery, the treatment of wounds of the heart and blood vessels was the springboard for further advances in cardiovascular disorders. It is generally agreed that wounds of the heart should be operated upon if there is continuing bleeding to the outside or into the pleural cavity. Fortunately, various advances in surgery, including improvements in anesthesia, have decreased the mortality rate when operation is necessary. There is some difference of opinion how the patient with uncomplicated acute cardiac tamponade without continuing bleeding should be treated. Dr. Ravitch and I[4] are of the opinion that open operation should be deferred until the effects of at least one pericardial aspiration have been determined. It has been our experience that the majority of patients will respond favorably to one or more aspirations, that bleeding into the pericardial cavity will not recur, and that an open operation will not be necessary. Elkin and Campbell[5] have recently reported on the effectiveness of aspiration in the treatment of cardiac tamponade.

3. Systemic Arteriovenous Fistula: The majority of such fistulae are traumatic in origin, and hence are considered in this group. The diagnosis and the treatment of traumatic systemic peripheral arteriovenous fistula are clear-cut. The mortality in the many patients operated upon during World War II was amazingly low. It is generally agreed that the best treatment consists of eradication of the fistula with repair of the openings in the artery and vein, and that the second method of choice is quadruple ligation of the artery and vein with excision of the fistula provided the continuity of the artery can be interrupted without the loss of distal parts. There is some difference of opinion as to the choice of time of operation. If the artery involved is one which can be spared if necessary, one may operate as soon as the fistula is discovered. If sacrifice of the involved artery would subject its part to the danger of gangrene, an interval of a few weeks should be allowed to elapse following the injury in order to permit the further development of collaterals.

Most of the congenital peripheral arteriovenous fistulae involve bone, and here the problem of treatment may be an exceedingly difficult one. If the bony involvement is limited,[6] one may excise the segment with or without replacement with a graft. Unfortunately, however, the lesions are usually extensive, and multiple amputation may be necessary.

The subject of intracranial arteriovenous fistulae is a difficult one with many unsolved problems and will not be discussed.

4. Arterial Aneurysm: With better control of syphilis, the incidence of aneurysms should lessen. On the other hand, there will probably be an increase in those due to arteriosclerosis unless preventive and therapeutic measures decrease the incidence of this disease. Unfortunately, the arteriosclerotic process is usually not limited to the aneurysm itself, and this fact renders it important in surgical treatment to interfere as little as possible with the blood supply of parts distal to the aneurysm. The Matas endoaneurysmorrhaphy, the wiring procedure as used by Finney and others and further advanced by Blakemore, and the placing of irritating cellophane around the sac have in common the advantage that they need not interfere unduly with the collateral arterial pathways around the aneurysm. This is particularly true of the wiring procedure. Blakemore[7] now treats some aneurysms by constricting the artery proximal and distal to the aneurysm with polythene tape, thereby reducing the pulse pressure, and by heating wire introduced into the sac. The relief of pain and the abolition of respiratory difficulty that may follow the wiring of a thoracic aneurysm is a dramatic accomplishment. If the results that are being obtained by Gross[8] and others on the homotransplantation of aortic grafts continue to be encouraging, it is likely that the procedure can be used in conjunction with excision of the aneurysmal sac when the neighboring segments of aorta are not grossly diseased. Lam[9] has inserted an aortic graft without excision of the sac. The development and use of improved plastic materials may be helpful in the treatment of aneurysms.

5. Mitral Stenosis: It may seem unduly optimistic to place this disorder in this group, but I do so because of the surprisingly good early results that are being obtained at present. Most cases of mitral stenosis are due to rheumatic fever, and it is probable that great advances will be made in the prevention or treatment of rheumatic infections. Even without further advances, it is likely that the use of antibiotic and other agents which are available will reduce the repeated attacks of infection and thereby lessen the incidence of mitral stenosis. On the other hand, many attacks of acute rheumatic fever are unsuspected or undiagnosed and the later development of some chronic disorder may give the first unmistakable clue to an earlier attack of an acute infection. For this reason it is likely that there will continue to be a moderate number of cases of mitral stenosis despite preventive and therapeutic advances.

Following the discouraging attempts[10, 11] which were made to treat mitral stenosis by surgical methods in the third decade of the present century, it is largely to the credit of the late Horace Smithy,[12] Bailey,[13], and Harken[14] that the problem was revived several years ago. In view of the present encouraging results, in some of which the procedure consists of dilation of the stenotic valve with the finger, it is difficult to understand why the successful use of this method by Souttar[15] in 1925 did not stimulate greater interest at the time. Whereas Cutler and his associates believed that a segment of the stenotic valve should be removed, and a similar view was held by Smithy, both Bailey and Harken are of the opinion that dilation of the valve and division or tearing of the commissures produce an opening of adequate size and that mitral insufficiency does not result. Both Bailey and Harken prefer an approach through the left auricular appendage as was advocated by Allen and Graham, and there is no doubt that this is preferable to that through the left ventricle. Bailey employs a knife which is attached to the index finger for division of the commissure region. Harken attempts to fracture or tear the commissure region with the index finger, and if this fails he divides it with a knife. It is our experience that in most of the cases one or both of the commissure regions can be torn with the index finger and that the use of a knife is generally unnecessary. Bailey does not favor the making of much pressure with the finger because of fear of tearing one of the valve leaflets. We have not as yet had the unfortunate experience of tearing one of these. It should be emphasized that it is not always possible to tear the commissure region and that one should have a proper cutting instrument available.

The early results that are being obtained in the treatment of mitral stenosis by direct attack on the valve are surprisingly good. The subjective improvement of the patient is usually greater than the alteration in the measurable dynamics of the circulation. It is too early as yet to know what the long term results will be, but even the improvements of the duration that have been recorded to date justify the operation. Subsequent observations may show that there is a *gradual reformation* of the stenosis. If such is the case, it may be advisable to remove a segment of the valve, the plan of attack which was used by Cutler, Graham, Smithy and their associates. Since there are still unsolved problems, it is important that efforts to produce mitral stenosis in experimental animals be continued.

In addition to recent progress in the direct attack on the stenotic mitral valve, efforts have been made to prevent bouts of pulmonary edema in this condition by the anastomosis of systemic and pulmonary veins as performed by Sweet[16] or the making of an interauricular defect as advised by Harken.[17] It is obvious that these shunting procedures should not be used if there is a very tight stenosis, indicated by small left ventricular output and inability to increase it with exercise, for a further reduction in output caused by the shunt will not be tolerated. There are probably some cases in which some such procedure will be helpful, but in general it would appear that the surgical treatment of mitral stenosis should consist of a direct attack on the stenotic area itself.

Much probably remains to be learned about the choice of patients in whom a direct attack on the mitral valve is indicated. According to Andrus, with whom I have been associated in this work, the indications for a direct attack on the stenotic mitral valve are as follows: Symptoms and signs of pulmonary engorgement—dyspnea and cough on exertion, paroxysmal dyspnea, pulmonary edema, or frank hemoptysis. The most promising cases are those without enlargement of the left ventricle or prominent enlargement of the left auricle; usually these are signs that mitral insufficiency contributes significantly to the patient's disability. In general, there should be predominantly a stenosis, although a small degree of insufficiency is not a contraindication to commissurotomy. If one uses fairly rigid criteria in the selection of patients, the mortality associated with operation is fairly low. One of the twenty-five patients upon whom we performed a commissurotomy died postoperatively. An additional patient who was found at exploratory thoracotomy to have an obliterated auricular appendage, no commissurotomy being performed, succumbed subsequently from a cerebral embolus.

B. Congenital Lesions.—

6. Patent Ductus Arteriosus: Although a procedure for the correction of patent ductus arteriosus had been suggested many years previously by Munro[18] and had been attempted by Strieder,[19] it is to the everlasting credit of Gross[20] that he was the first to close a patent ductus successfully. The typical case is not difficult to diagnose, and technical procedures are available for obtaining a permanent closure of the ductus with a low mortality. In view of these facts, and because of the dangers of allowing a ductus to remain patent, it is my belief that operation is indicated for all children beyond two or three years of age who have an obviously patent ductus arteriosus. However, it is often difficult to be certain of the diagnosis in infants. Furthermore, Dammann[21] and Keith[22] have recently called attention to the fact that some patients with a very large patent ductus and associated pulmonary hypertension may not have a typical machinery-like murmur. In some of these patients the presence of a large patent ductus has been confirmed by thoracotomy. There is some difference of opinion whether one should divide the ductus and close the two ends as preferred by Gross[23] and others, or whether one should close the undivided ductus by the use of multiple suture ligatures placed as far apart as possible. My associates and I prefer the multiple suture technique[24] because it seems to us that it is easier and safer. The desideratum is permanent closure of the ductus, and we are aware of only one instance of recanalization in the approximately 300 patients in whom we have used this method.[25] The exception was a child of five who had a ductus which measured 16 mm. in diameter.

Whereas the operation for closure of a patent ductus is not a very dangerous one in most children and young adults, the procedure becomes more difficult and dangerous in the older age groups. Pulmonary hypertension with disease of the walls of the pulmonary artery and ductus increases the likelihood of uncontrollable bleeding from these structures. In the older patients with an enlarged heart and pulmonary hypertension we use a long posterolateral incision instead of the anterolateral incision which is preferred in the less complicated cases. In the occasional patient with extreme pulmonary hypertension in association with a patent ductus there is some flow of blood from the pulmonary artery to the aorta. This reverse flow of blood is particularly apt to occur if the patent ductus is attached to the aorta distal to a coarctation of the aorta. Closure of the ductus is especially dangerous in patients with severe pulmonary hypertension and a reversal of flow through the ductus. In addition to the hazards of hemorrhage, there is the possibility that closure of the ductus will result in a sudden increase in the diastolic volume of the right ventricle and undue distention of that chamber.

Now that the diagnosis of the patent ductus can be made in most instances with accuracy and the operative technique is perfected, it is to be hoped that the condition will be recognized and treated in childhood and that in the future fewer cases will be encountered in adults.

An aortic septal defect providing a communication between the aorta and the pulmonary artery may give signs and symptoms similar to those of the usual patent ductus. A small aortic septal defect may be closed by existing techniques, but the large one extending to the base of the heart presents problems that have not been satisfactorily solved.

7. *Coarctation of the Aorta:* Fortunately there is usually no difficulty in the diagnosis of coarctation of the aorta. If there is any reason to suspect that the coarctation is at an unusual site, angiocardiography or aortography should be used in demonstrating the lesion. It is not necessary to use these radiological methods in the majority of cases.

The proper treatment of the adult type of coarctation, the common form in which the constricted segment is short, is *essentially that which was first* employed successfully by Crafoord,[26] and consists of excision of the stenotic or atretic segment and an end-to-end anastomosis of the aorta. It is desirable to preserve the dilated intercostal arteries. There is some difference of opinion as to the type of suture that should be used in making the anastomosis. We usually employ a continuous everting suture of silk which is interrupted at several points. An overhand noneverting suture also gives satisfactory results. Interrupted sutures probably cause the least disturbance of growth of the anastomotic site and should be used in young children. Several recent experimental studies indicate that the size of the lumen at the site of the anastomosis increases somewhat with the growth of the subject even though a continuous suture is used. This brings up the question as to the age period in which the operation should be performed. I think the ideal period is from six to fifteen years of age. Complications in connection with coarctation rarely occur before the age of six, although in one of our patients hemiplegia developed at the age of two years. The aorta of a child of six is usually of sufficient size to provide a good lumen following anastomosis even though subsequent growth of this site may not be great. The employment of interrupted sutures will increase the likelihood of growth. I have no strong convictions as to the upper age limit at which operation should be advised. We have operated upon a number of patients in their thirties and one in the early forties with good results. On the other hand, I am inclined to think that a patient past forty who has no serious symptoms or complications save for the hypertension accompanying the coarctation should not be operated upon.

The treatment of the infantile type of coarctation in which the constricted segment is long presents a more difficult problem. The same may be said for the occasional case of the adult type when the proximal or distal segment is hypoplastic for a short distance. I think a good result will be obtained in most cases by the use of the left subclavian artery[27] to bridge the narrowed segment if the procedure is performed properly. This means that the constricted segment should be excised in toto, and the proximal end of the aortic segment should be closed with an arterial suture; the left subclavian artery should be cut across proximal to its initial branching, and should then be rotated downwards in such manner that there is no sharp twisting or angulation, and its end should be anastomosed to the distal end of the aorta. If one performs the operation without removing the constricted aortic segment and closing the proximal end of the aorta and without proper rotation of the subclavian, a good straight channel for the flow of blood is not obtained. Usually the subclavian artery is almost as large as the distal aorta, and there is little difficulty in performing a good union. The other alternative is the employment of a homograft of aorta as used by Gross[8] to replace the constricted segment of the aorta. The early results in the experience of Gross have been amazingly good. It is too early as yet to know how the homograft will weather the passing of time. The chances are that it is replaced by the recipient's own tissues. Disadvantages of this procedure are that it is difficult to obtain proper grafts and that two arterial anastomoses have to be performed. The severe constriction of the aorta that is present in the infantile type of coarctation should usually be discovered in childhood, and the use of either the transposed subclavian artery as a substitute for the segment or the employment of homografts should give fair results. Unfortunately, the stenosis may be proximal to the origin of the left subclavian artery and may be associated with other congenital defects.

The question remains unsettled why some patients who are operated upon for the relief of coarctation do not have the desired decline in blood pressure. Fortunately, this is the exception rather than the rule. Among the possibilities is the fact that even after operation the aortic lumen in some patients is still not as large as it should be. This is particularly likely to be the case when the lumen of the proximal aortic segment is smaller than normal. Another possibility, if the kidney is involved in this type of hypertension, is that renal changes may persist after operation. Still another possibility is that irreversible alterations in the arterioles may have occurred in connection with the hypertension. I think that the number of disappointing results will decrease as additional experience is gained by the performance of larger and more perfect anastomoses. We are fairly well pleased with the results of operations on 104 patients with coarctation.

8. Pulmonary Stenosis and Atresia: The most frequent example of this type is the typical tetralogy of Fallot.[28] Other types of malformations which may be associated with pulmonary stenosis or atresia include tricuspid atresia, single ventricle, pseudotruncus arteriosus, dextrocardia or dextrorotation of the heart, situs inversus viscerum with levorotation, and valvular pulmonary stenosis with intact ventricular septum.

The typical case of *tetralogy of Fallot* presents few difficulties in diagnosis. There is need for more accurate appraisal of the degree of overriding of the aorta, the length of the infundibular stenosis, and the size of the infundibular chamber since this information may alter treatment, as will be discussed later. Patients with excessive dextroposition of the aorta may not do well following the creation of an artificial ductus. With the increase in the blood flow to the lungs there will obviously be an increase in the amount of blood returning to the left side of the heart. Since in cases of extreme dextroposition the aorta receives blood mainly from the right ventricle unless the interventricular defect is large, the left ventricle may be unable to empty itself properly and pulmonary engorgement and edema may develop. One possible means whereby the degree of overriding might be determined more accurately is by the injection of radiopaque material through a catheter placed in the right ventricle and the taking of repeated roentgenograms. The possible value of this procedure will be determined only by multiple trials. If an extreme degree of overriding were proved to be present, one would certainly not wish to create a large artificial ductus. Perhaps some day a safe means will be available for increasing the size of the interventricular defect. This could be done at the same time that the ductus is made.

It is largely as a result of the observations of Brock[29] that the efforts toward a direct attack on infundibular stenosis rather than the creation of an artificial ductus have been stimulated. The direct attack is preferable to the anastomotic procedure, on theoretical grounds at least, because it reduces the shunt through the interventricular defect at the same time that it increases the pulmonary flow, and it does not add another anomaly to those already present.

Some idea as to the size of the infundibular chamber and the length of the stenosis may be obtained by angiocardiography. The information might be more exact if the diodrast were injected through a catheter introduced into the right ventricle. In addition to the information gained in preoperative studies, one can obtain some idea as to the size of the infundibular chamber by exposing this region at the time of operation. There are certainly many cases of the tetralogy of Fallot in which there is a long infundibular stenosis and in which a direct attack on the stenosis is not feasible. There are probably a moderate number of cases with a short diaphragm-like zone of stenosis and a large infundibular chamber in which a direct attack is possible. As indicated previously, there is need for more accurate means for determining the exact condition of the lesion. In some patients with a short infundibular stenosis it is not difficult to introduce dilating and cutting instruments through an incision in the right ventricular wall and through the stenotic area, as we have done in a few cases following the lead of Brock. It is much more difficult and dangerous to excise part of the stenotic area as performed by Brock[30] and Glover,[31] and others. Technical improvements are needed in this respect. Furthermore, the question remains unsolved whether such a stenotic area in the heart muscle which is treated by dilation and incision or excision will subsequently reform. It is my opinion that improvements in accuracy of anatomic diagnosis and in the technique of intracardiac procedures will result in an increasing number of direct attacks on infundibular stenosis. At the same time Dr. Taussig and I believe that the majority of these patients will have to be treated by the creation of an artificial ductus. Certainly this latter procedure rather than a direct attack offers hope of improving the patient with pulmonary atresia. In this connection it should be mentioned that simple exploratory thoracotomy with the subsequent formation of adhesions and the resulting additional collateral blood supply to the lungs seems to improve the condition of some patients in whom the pulmonary artery is not of suitable size for an anastomosis. The number of collateral channels is probably increased by removing the parietal pleura as recommended by Barrett.[32]

The techniques that are available for the creation of an artificial ductus are well developed, and the results are amazingly good in the typical tetralogy of Fallot. Dr. Taussig[33] is of the opinion that the occasional patient who does not do well following a satisfactory anastomosis does not have an uncomplicated tetralogy of Fallot. We perform an end-to-side, an end-to-end or a side-to-side anastomosis between a systemic and a pulmonary artery, the choice depending upon the age and size of the patient and the observations at the time of operation. This choice has been considered in detail in a previous publication.[34] In the 20 odd per cent of patients in whom the aorta descends on the right, the incision is almost invariably made on the left, and an anastomosis performed between the left subclavian and the left pulmonary artery.

In patients in whom the aorta descends on the left, the incision is usually made on the left in infants, in adolescents, and in adults, and it is usually made on the right in children between the ages of two and twelve years. In patients in whom there is considerable doubt as to the diagnosis, for example when valvular pulmonic stenosis is suspected, the incision is made on the left in order that one may examine the structures at the base of the heart. When the incision is made on the left in the presence of a left descending aorta, the left subclavian artery is used for the anastomosis if it is of adequate size and length; otherwise the aortic side-to-side anastomosis as devised by Potts[35] is employed. We do not hesitate to use an end-to-end anastomosis between the subclavian artery and the divided distal end of one of the pulmonary arteries if the pulmonary artery is unusually small or if the condition of the patient is poor and one wishes to perform the procedure speedily and without traction on the mediastinum. Dr. Taussig and I consider the optimal time for operation to be between the ages of five and ten years. A second operation at a later period is less apt to be necessary if the child has reached the age of five or more before the initial operation is performed. Unfortunately, the condition of many infants and young children is such that earlier operation is required. The general philosophy of Dr. Taussig's cardiac clinic is, "The sicker the sooner."

The results of the creation of an artificial ductus in the treatment of pulmonary stenosis or atresia are not so good when there are associated malformations such as tricuspid atresia, single ventricle, dextrocardia or dextrorotation, or situs inversus with levorotation, as they are in the typical uncomplicated tetralogy. Tricuspid atresia is usually associated with a diminutive or absent right ventricle and some abnormality of the pulmonary orifice. Some patients with this condition are improved by the creation of an artificial ductus. Others do poorly, due in part at least to a resulting increase in strain on the left ventricle in the absence of an interventricular defect. Patients with tricuspid atresia and with only a small opening between the two auricles may be improved by the creation of a large interauricular defect. If one employs the method which Hanlon and I[36] devised for this purpose, one has to be very careful during the procedure not to occlude entirely the existing communication between the two auricles for more than a few seconds. Patients with a single ventricle respond very poorly to the creation of an artificial ductus, and the operation should not be advised if the diagnosis can be made and if the patient is getting along moderately well. Patients with dextrocardia, or dextrorotation of the heart, or situs inversus viscerum with levocardia are likely to have a variety of malformations and are likely to respond unfavorably to the creation of an artificial ductus.[37]

Valvular pulmonary stenosis with or without a communication between the two auricles and with an intact ventricular septum occurs approximately 10 per cent as often as the tetralogy of Fallot. The work of Brock,[29] of Sellors,[38] and of others has added materially to the diagnosis and treatment of this condition. Cyanosis is present in about 70 per cent of the cases and is due to a right to left anastomotic procedure, on theoretical grounds at least, because it reduces the shunt through the interventricular defect at the same time that it increases the pulmonary flow, and it does not add another anomaly to those already present.

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pulmonic stenosis is only moderately thickened, and I believe that incision and dilation will result in a permanent increase in the size of the opening. It appears likely that endothelium grows over the incised area and that the opening remains patent. If such should prove not to be the case, it will be advisable to remove part of the valve as has been performed by Varco.[41] No ill effects attributable to pulmonary insufficiency have been observed in any of our patients subjected to pulmonary valvulotomy, and it would seem that patients should withstand removal of part of the valve without ill effects.

9. Pulmonary Arteriovenous Fistula: This condition is being diagnosed with increased frequency and should be suspected in cyanotic patients in whom there is no evidence of disease of the heart itself. In doubtful cases, angiocardiography is helpful in diagnosis. Evidence of abnormal communications between pulmonary arteries and veins may be late in developing. Unfortunately, the communications are usually multiple and parts of both lungs may be affected. Treatment consists of excision of the diseased areas; in some instances a wedge-shaped resection will suffice. If a lobe is extensively involved it should be removed. One should not, however, remove an entire lung unless there are no normal segments present for fear that evidence of abnormal arteriovenous communications will subsequently appear in the remaining lung. One of the most dramatic results which I have witnessed in surgery followed the removal of a lobe of the lung containing multiple arteriovenous communications from a 28-year-old woman. Total incapacity was replaced by perfect health. The saturation of the arterial blood with oxygen is now normal and there has been a gain of 36 pounds in weight.

10. Anomalies of the Aortic Arch: Anomalies of the aortic arch and its major branches occur frequently in patients with congenital heart disease, but only a few of these in themselves cause any difficulty. A small proportion of these anomalies interfere with breathing or swallowing or both due to pressure on the trachea and esophagus. This difficulty is particularly likely to be present if there is a bifid aorta, although the same symptoms may be caused occasionally by a retro-esophageal artery such as an aberrant subclavian. Neuhauser[42] has made important contributions to the diagnosis of this condition. Gross[43] and others have shown that the treatment consists of the division of the offending blood vessel. In the case of the bifid aorta, the smaller of the two arches should be divided and the two ends closed. The most interesting patient with a bifid aorta in our experience is a boy who had tetralogy of Fallot as well. Treatment consisted of division of the smaller arch and an anastomosis of its distal end to the left pulmonary artery. In general, surgical treatment of anomalies of the arch is satisfactory.

II. DISORDERS IN THE TREATMENT OF WHICH SURGERY MAY BRING ABOUT MODERATE IMPROVEMENT

A. Acquired Lesions.—

11. Essential Hypertension: Whereas there is a high percentage of good results in the treatment of hypertension associated with coarctation of the shunt through a patent foramen ovale or an auricular septal defect. The pooling of diodrast as observed on angiocardiography in the pulmonary artery distal to the valvular stenosis is an important clue in diagnosis. The pressure in the right ventricle is significantly elevated and may exceed that in the left ventricle. Bing recorded a right ventricular pressure of 223 mm. Hg in one of our patients. The conditions observed at the time of operation are characteristic. There is definite right ventricular hypertrophy, usually a poststenotic dilatation of the pulmonary artery, a jet of blood can be felt striking the wall of the pulmonary artery, and usually the dome-shaped pulmonary valve can be palpated. Patients with valvular pulmonary stenosis are not improved by the creation of an artificial ductus. It is true that cyanosis if present is lessened, but these patients subsequently have right-sided heart failure.

We have followed essentially the technique of Brock in treating valvular pulmonary stenosis.[39] The approach is through an incision in the right ventricle, and the valve is cut and dilated. The Brock flat valvulotome with a probelike end and two sharp cutting edges is a very satisfactory instrument. Even so, the umbrellalike instruments recently developed by Potts present some advantages. In general we have been greatly pleased with the results obtained but a number of questions are as yet unanswered. Among these are the following: (1) Why is it that the blood pressure usually declines and the heart may fail shortly after opening the chest and exposing the heart? (2) Why is it that the pressure in the right ventricle may not fall to normal following division and dilation of the valve and in the presence of definite improvement in the condition of the patient? (3) Should operation be advised prior to the development of great incapacity and of right ventricular hypertrophy and dilatation? (4) Will extreme right ventricular hypertrophy, once developed, ever disappear entirely following operation? (5) Should the stenotic valve simply be incised and dilated as is done at present, or should a segment of the valve be excised? (6) When the valve is incised and dilated, do the incisions in the leaflets remain open or do they partially grow back together?

A longer postoperative follow-up of the patients and a repetition of the studies will probable supply an answer to some of the above questions. It is known that patients with great enlargement of the heart are those in whom difficulty is most likely to be encountered during the operation, and I believe that patients should be operated upon in advance of this enlargement if the condition is diagnosed. It has been observed in some of our patients that the size of the right side of the heart has decreased postoperatively, but this may be due to a disappearance of the dilatation rather than to a regression of the hypertrophy. The finding of Dr. Bing[40] that the pressure in the right ventricle usually does not fall to normal in the first few months after operation may be explained on the basis of inadequate division and dilation of the valve or possibly on the basis of partial obstruction at the pulmonary orifice by the greatly hypertrophied right ventricular muscle. I have already commented on the fact that patients show decided improvement postoperatively despite the persistence of some right ventricular hypertension. The valve in congenital heart and lung. Even if this proves to be feasible ultimately, there is still the problem that the coronary arteries could not be transposed and that they would continue to receive venous blood from the right ventricle. Almost certainly the oxygen content of this blood would be higher than before the positions of the aorta and pulmonary artery were corrected, and it is possible that it would suffice for the nourishment of the myocardium. On the other hand, it is known that infants with an anomalous origin of coronary arteries from the pulmonary artery usually do poorly. However, the pulmonary arterial pressure in these patients is low whereas it is high in those with transposition. Even if it becomes technically feasible to correct the position of the aorta and the pulmonary artery except for their orifices, and even if the myocardium should receive adequate oxygen from venous blood, there still remains the problem of the pulmonary hypertension. It is possible but not at all certain that it would disappear after correction of the position of the great arteries.

Since one could not alter the position of the coronary arteries even if one were able to correct the positions of the aorta and the pulmonary artery, perhaps the ideal would be the development of methods whereby the systemic and pulmonary veins could be transposed, leaving the aorta and the pulmonary artery in their transposed positions. If total transposition of the venous return could be accomplished successfully it would probably result in great improvement in the condition of the patient. The coronary arteries would receive arterial blood, and one's greatest concern would be with what would happen to the pulmonary hypertension. Unfortunately, an experimental complete transposition of the systemic and pulmonary veins is difficult if not impossible with existing techniques. Brown and I[47] have accomplished in the experimental animal a transposition of the pulmonary venous return, but this required the removal of most of the left lung and the making of a large interauricular defect. Anastomoses of veins to the auricular appendage or auricle are very likely to become occluded. Anatomic relationships add to the difficulties of producing complete transposition of veins, such as, for example, the fact that the inferior vena cava is some distance from the left auricular appendage. Transposition of some of the pulmonary veins can be produced without too much difficulty, and we used this method to a limited extent in our early efforts to treat transposition in patients. Unfortunately patients with transposition of the great arteries tolerate a partial alteration in venous return very poorly since it causes an imbalance between the two sides of the heart. The situation is totally different from that existing in the normal heart where the blood leaves from one side and returns to the other.

After this lengthy discussion it should be repeated that our best results in the treatment of transposition of the aorta and pulmonary artery have followed the creation of a large interauricular defect. We are encouraged but are not satisfied with the results, and better methods should be devised.

13. Anomalies of Venous Return: These anomalies may occur in either the pulmonary or the systemic circulation. In other words, some or all of the pulmonary veins may empty into the right auricle or into veins that drain into the right auricle, and part or all of the blood returning through the venae cavae may empty into the left auricle.

If all the pulmonary veins drain into the right auricle or its tributaries, there must be an interauricular defect to make the condition compatible with life. In most patients in whom several but not all of the pulmonary veins drain into the right auricle or its tributaries there are associated malformations. In one of the patients studied by Dr. Bing[18] in whom several but not all of the pulmonary veins emptied into the superior vena cava but in whom there were no associated malformations, there was a great increase in pulmonary blood flow without an associated increase in the pulmonary pressure. The ideal in surgical therapy in such a case would be the anastomosis of the aberrant veins to the left auricular appendage or one of its tributaries. Almost equally good results would probably be accomplished by the less difficult procedure of making a large interauricular defect. Muller[19] has recently anastomosed the side of an anomalous left pulmonary vein to the left auricular appendage with a good result. Dr. Bing has found pronounced pulmonary arterial hypertension with strain on the right ventricle in two patients in whom all of the pulmonary veins drained into the right auricle through the superior vena cava. The systemic arterial oxygen saturation in these patients was only moderately depressed, and the main problem was the burden on the right ventricle. An attempt was made in one of these patients to correct the venous return but without success. The creation of a very large interauricular defect would probably have relieved the strain on the right ventricle. The larger the auricular defect the greater will be the opportunity for a right to left shunt.

As pointed out earlier, there is danger of spontaneous closure of an anastomosis between a vein and an auricle or auricular appendage. The anatomic structure is such that it is difficult if not impossible to anastomose the right pulmonary veins to the left side of the heart unless they actually pass across the midline and into the left chest. In some instances all of the pulmonary veins, right and left, do come together in the left side of the chest and enter the innominate vein or the superior vena cava by a common trunk. Under such circumstances it is important to realize that one is dealing with a common trunk and that total occlusion while one is performing an anastomosis to the auricular appendage would result fatally. If the trunk is large, it may be partially occluded while the anastomosis is being performed; otherwise one of its branches may be used for this purpose. As indicated previously, the implantation of the anomalous vein into the correct side of the heart is the ideal procedure, but the dangers of subsequent closure of the anastomosis and the difficulties of determining the exact anatomic picture may influence one to make a large interauricular defect if one is not already present.

Important anomalies of the venae cavae are less frequently observed. If an anomaly is discovered that is producing symptoms, the problems in treatment are similar to those that have been discussed in connection with the pulmonary veins; that is, transplantation into the proper side of the heart or the making of a large interauricular defect. Bilateral superior venae cavae occur fairly frequently and are usually associated with other malformations. Usually both of the superior venae cavae enter the right side of the heart, but in some instances the left one drains into the left side. If this latter condition as well as the presence of a normal right superior vena cava is determined preoperatively, one may ligate the left superior vena cava in the course of attacking the associated congenital malformation.

III. DISORDERS IN THE TREATMENT OF WHICH SURGERY IS OF DOUBTFUL VALUE OR SUITABLE METHODS ARE NOT YET FULLY DEVELOPED

A. Acquired.—

14. Coronary Artery Disease: I hope that I am in error in placing this disorder in this group because if it is proved that many cases of coronary arterial disease are amenable to surgical therapy, it will constitute one of the greatest of all advances in medicine. However, it would appear that our chief hope lies in the prevention of arteriosclerosis, possibly by dietary measures.

It is largely due to the ingenuity and tenacity of Dr. Claude Beck[50, 51] that some surgical progress has been made in this most difficult field. In many experiments and some clinical trials, efforts have been made to protect the heart against coronary occlusion by the establishment of an extracoronary bed and of communications between the three main coronary arteries. The methods which have been employed include the grafting of adjacent tissues such as skeletal muscle, lung,[52] pericardium, and omentum onto the surface of the heart in an effort to supply blood to the ischemic myocardium from an outside source; the use of chemical or physical inflammatory agents applied directly to the heart in an effort to stimulate intercoronary communications; and more recently attempts to cause a backward flow of blood through the coronary veins and capillaries by a direct anastomosis between a systemic artery and the coronary sinus of the heart.[51, 53] Other efforts which have been made to improve the condition of the patient with coronary arterial disease and angina pectoris include thoracic sympathectomy for the relief of pain, ligation of the great cardiac veins, which may cause stasis and a better distribution of blood in the myocardium, total thyroidectomy, the implantation of a systemic artery into the myocardium, and the excision of infarcts of the heart.

The operation which appears to me to have the most likely possibilities for improving the circulation of the myocardium is the original one proposed by Beck[50] in which some tissue adjacent to the heart is placed against the heart and an inflammatory reaction is provoked by an agent such as talc. In addition to the fact that this is a less dangerous procedure than that in which an anastomosis between a systemic artery and the coronary sinus is created, it would seem that the chances for prolonged improvement in the circulation are greater. Whether one uses total, partial, or no occlusion of the coronary sinus at its ostium in conjunction with its anastomosis to a systemic artery, it is likely that the shunted arterial blood will ultimately find its way into the heart chambers through dilated venous channels in which the pressure is lower than that in the capillaries.[54, 55] I am skeptical of the value of the systemic artery to the coronary sinus operation, despite the fact that Heimbecker and I[56] have shown recently by observation of capillaries and determinations of the oxygen content of blood that arterial blood can be made to flow backward through the veins and capillaries of the intestinal tract, for a brief period at least, under conditions which will not be detailed here. Fluids seek a low pressure route and blood will not flow through a high pressure zone for an extended period when small veins exist which are capable of dilation when subjected to increased pressure.

Beck has emphasized his belief that the operations which he has described will not eradicate the disease and that a complete cure is not to be expected. Since the average duration of life of patients who survive the first attack of coronary occlusion is approximately five years, it is extremely difficult to evaluate the effects of a procedure which is not curative, and thousands of trials will be required before a definite answer can be supplied. Even though it would appear that the possibilities of making real progress in the treatment of coronary disease by existing techniques are not great, the importance of the problem demands that experimentation be continued.

15. Aortic Valvular Stenosis: Aortic stenosis as an isolated lesion is rare and is more often acquired than congenital. Rheumatic fever is the most frequent causative agent. It is fortunate that the lesion is less common than mitral stenosis, for the surgical problem is thus far a more difficult one. Our efforts to treat aortic stenosis are limited to two cases and both resulted fatally from ventricular fibrillation. The first patient (who was to be operated upon by the late Horace Smithy) died as the incision was being made in the chest wall, and the second died shortly after the stenotic valve had been incised and dilated through an incision in the left ventricle. Bailey[31] has attempted to stretch the stenotic orifice by the use of a dilator introduced through the carotid artery and down the aorta, but he has now abandoned this method in favor of the ventricular approach.

Subaortic stenosis,[57] which is also rare, is usually a congenital lesion and is caused by the persistence of a band or membrane of connective tissue which lies immediately below the aortic valves. The problem of surgical therapy should be easier in this condition than in rheumatic aortic stenosis.

16. Insufficiency of Heart Valves: The problem of the surgical treatment of insufficiency of the various valves of the heart is still decidedly in the experimental stage and will probably remain there for some time. One could probably anchor in place an artificial mechanical valve by the use of existing techniques, but there is great doubt whether such a valve would function for an extended period. If one is to place accurately a graft of viable tissue such as pericardium, one should be able to suture with care in a dry field under direct vision, which means that an extracorporeal circulation is required. Furthermore, it remains to be determined whether such valves will live and function properly. This latter problem may present greater difficulties than those of artificial circulation. Gordon Murray and his associates[58] used vein segments, and Templeton and Gibbon[59] employed vein and pericardial grafts in efforts to repair experimental defects in valves. The results are moderately encouraging, particularly with the use of pericardium, but the observation period is too short for critical assessment. Even if the results in partial replacement of the normal valve by a graft were practically perfect, the question is still unanswered as to the fate of a graft placed in a scarred area such as exists in an acquired insufficiency. The functioning of heart valves is an extremely complicated mechanism and is dependent upon the attachment of the chordae tendinae as well as other factors. At any rate, the problem of valvular insufficiency is an important one which it will be very difficult to solve.

I am aware of the fact that Bailey and his associates are placing a pedicled flap of pericardium across the left ventricle just below the mitral valve in the treatment of mitral insufficiency, and it is said that the early results are encouraging. A long observation period will be required for proper evaluation.

17. Intracardiac Tumors: Existing surgical methods are suitable for the excision of tumors of the surface of the heart. Unfortunately these lesions are usually metastatic. The proper treatment of tumors of the interior of the heart awaits the development of more accurate means of diagnosis as well as means either for good visualization with instrumentation of the interior of the beating heart, or perfection of the artificial circulation which will permit an open operation in a bloodless chamber of the heart.

B. Congenital.—

18. Anomalous Origin of Left Coronary Artery: In the rare anomaly in which the left coronary artery arises from the pulmonary artery the infants usually die of cardiac failure during the early months of life. This condition may occur as the sole anomaly. Bland, White, and Garland[60] have shown that the condition can sometimes be diagnosed by the finding by electrocardiography of inversion of the T waves in all three leads combined with low voltage curves.

There is a possibility but not a great likelihood that this condition may be treated surgically by severing the connection of the left coronary with the pulmonary artery and anastomosing its distal end to a systemic artery such as the subclavian. It would be necessary to perfuse the coronary artery with blood while the procedure is being performed. Smith and I have made a few attempts to carry out a similar procedure in experimental animals but thus far without success. The small size of the coronary artery in an infant renders perfusion and simultaneous anastomosis a difficult and dangerous undertaking.

19. Auricular Septal Defects: A defect of the auricular septum is a common lesion and frequently an isolated one. These defects vary greatly in size and position. The shunt of blood is usually from the left to the right auricle and hence there is usually no cyanosis. The shunt imposes an added load on the right side of the heart, and there is generally dilatation of the right atrium and ventricle and right ventricular hypertrophy. Failure of the right side of the heart may occur. In some cases the shunt is reversed, that is, from right to left, and cyanosis is present. In the absence of pulmonary hypertension it seems obvious that a more balanced circulatory pattern would result from closure of an isolated auricular septal defect.

When pulmonary hypertension is present it may very well be that closure of an interauricular defect is contraindicated. The answer probably hinges on the question whether the pulmonary hypertension is primary or secondary, and if secondary, whether the hypertension would be lessened by closure of the defect. In the presence of marked arteriolar disease in the lungs the right ventricular pressure and consequently the right auricular pressure are elevated. As a result the shunt in the two auricles may be directed from right to left. Under these circumstances repair of the auricular defect would close the opening which may be preventing an excessive increase in pressure in the right auricle and may transform the condition into one resembling Ayerza's disease. Since the cause of pulmonary hypertension in association with an auricular septal defect is obscure and may be due to primary changes in the pulmonary arteries, I simply wish to emphasize the possibility that closure of the defect may be contraindicated. It is possible that closure of the defect combined with denervation of the lungs may be the treatment of choice. Denervation of the lungs without closure of the defect might make the condition worse.

Since there are many auricular septal defects unassociated with complicating factors, it is important that a satisfactory method for closing them be developed. Much experimental work along this line is being performed, such as that of Cohn[61] on the use of part of the wall of the auricle, and that of Swan[62] on the employment of the auricular appendages. One of the difficulties of the problem is that experimentally produced defects are prone to spontaneous closure, and it is difficult to gauge the effectiveness of various methods of repair. The method which Hanlon and I[36] devised for creating an interauricular defect usually results in one which does not close spontaneously, but its position is not the same as that of naturally occurring interauricular defects. Gordon Murray[63] made a preliminary report on his attempts to close interauricular defects by a blind method in which coarse suture material is passed through the heart in the region of the septum and is tied loosely. A somewhat similar method has been reported by Perianes,[64] I doubt whether any of the clinical or experimental methods which have been reported thus far will prove to be the answer to this problem. Good visualization of the interior of the auricles is desirable since the defects vary so greatly in size and position, and a wholly satisfactory method may have to await the perfection of the extracorporeal circulation.

20. The Lutembacher Syndrome: This syndrome consists of an auricular septal defect combined with congenital or acquired mitral stenosis and great dilatation of the pulmonary artery. It is well known that patients with auricular septal defects are prone to have rheumatic fever and mitral stenosis. The greatest strain in the Lutembacher syndrome is probably on the right auricle, the right ventricle, and the pulmonary artery, whereas the left ventricle and the aorta are spared except in the later stages. Because of the fact that the shunt is from left to right, there is usually no cyanosis. White[65] has pointed out that some patients with this condition survive to old age, but this is the exception rather than the rule.

If surgical therapy is indicated, it would appear that the attack should be on the stenotic mitral valve rather than upon the auricular septal defect. If the flow of blood through the stenosed mitral valve is increased by commissurotomy, the left to right shunt would decrease, thus lessening the burden on the right side of the heart. If the mitral stenosis can be dealt with effectively, and if pulmonary arterial disease is not present, it would probably be desirable to close the interauricular defect if a safe and suitable method were available. In the presence of pulmonary arteriolar disease the defect should not be closed even if the mitral stenosis has been alleviated since the defect probably serves as a safety outlet in permitting some right to left shunt and prevents an excessive rise in pressure in the right auricle. As stated previously, the pulmonary artery in the Lutembacher syndrome is greatly enlarged. Even if one could reduce the size of this vessel by surrounding it with an irritating material such as cellophane, it is doubtful whether the patient would be benefited.

Approximately one year ago I performed a mitral commissurotomy on a patient ill with the Lutembacher syndrome. She survived the procedure, but her condition is little if any improved at the present time.

21. Ventricular Septal Defects: Defects of the ventricular septum are frequently observed lesions, they are difficult to diagnose with certainty, they vary greatly as to size, location, and associated symptoms if any, and many of them are compatible with long life. The shunt of blood is usually from left to right. Since this shunted blood returns to the left side of the heart, both ventricles take part in transporting it. In this respect the situation is different from that in interauricular defects with a left to right shunt in that the right ventricle has the entire burden of pumping the shunted blood.

The prognosis in patients with small ventricular septal defects is excellent, and surgical therapy is not indicated. It is in patients with large high defects in whom the large shunt causes changes in the pulmonary blood vessels and in those in whom for some reason a right to left shunt develops that closure of the defect might be considered. In patients in whom a large shunt is predominantly left to right, closure of the shunt would undoubtedly benefit the patient by protecting the pulmonary vessels and reducing the strain on the heart. On the other hand, if pulmonary hypertension and a right to left shunt are present, closure of the defect might result in an increase in the residual volume of the right ventricle, a rise in pressure in the right ventricle and pulmonary artery, and in right-sided heart failure.

Ventricular septal defects are hard to produce experimentally and are likely to close spontaneously, which makes difficult the problems of surgical methods of closure. As far as I know, no satisfactory method has been developed. Murray[62] made a preliminary report of his efforts to close ventricular septal defects in patients by a blind suture method. Because of the fact that the exact position of the defect varies widely from patient to patient and that some of the defects are in close proximity to heart valves and to the orifices of major arteries, I do not believe that a blind method of suture is the answer to this surgical problem. The defects which produce symptoms are those that are closest to the heart valves and great arteries, and these present the chief difficulty in closure. As indicated previously, the low ventricular defect is a relatively benign lesion and closure is rarely if ever indicated. I am fairly certain that the proper surgical closure of ventricular septal defects that are producing symptoms will have to await the further development of methods of carrying on the circulation artificially which will allow under direct vision the careful placing and suturing of a graft of tissue such as pericardium in a position where there will be no interference with the action of heart valves or blockage of the orifices of important vessels. These technical difficulties added to those of accurate anatomic diagnosis indicate that it will be some time before this problem is solved.

22. The Eisenmenger Complex: In the Eisenmenger complex there is a high ventricular septal defect; the aorta overrides this defect and thus receives blood from both the right and the left ventricle. It differs from the tetralogy of Fallot mainly in that there is no pulmonic stenosis. The pulmonary artery is usually very large, and pressure within it is high. This condition, which fortunately is not common, may not be detected in childhood since cyanosis may not appear until the time of adolescence or later.

Serious changes in the pulmonary arterioles have been described in the Eisenmenger complex. Old and Russel[66] found the following: (1) medial hypertrophy; (2) an increase in elastic tissue with respect to both thickening of the pre-existing fibers and the presence of new fibers between the elements of the media; and (3) nonatheromatous intimal thickening with proliferation of fibrous tissue. These changes result in varying degrees of narrowing of the smaller pulmonary vessels. It has not been determined with certainly whether the pulmonary hypertension is primary or secondary, but it is very likely that it is secondary to the large high ventricular defect. If it were not for this increase in the pulmonary vascular resistance, most of the output of both ventricles would go to the lungs and the systemic flow would be inadequate. According to Bing,[67] the blood flow through the lungs in the Eisenmenger complex is essentially normal in quantity.

The ideal operation for the treatment of the Eisenmenger complex would be the closure of the high defect before pronounced and irreversible alterations in the pulmonary arterioles occur. As indicated in the discussion of the treatment of other high interventricular defects, this operation should be performed in a dry field under direct vision, and a graft of some tissue such as pericardium should be used. The proximity of the aorta to the defect renders an attempt at blind closure too dangerous. After definite pulmonary hypertension and visible cyanosis have developed, closure of the defect might result in failure of the right side of the heart. Since the pulmonary blood flow in this condition is said to be normal, and since there is severe constriction of the smaller pulmonary vessels, it is doubtful whether an artificial reduction in the size of the pulmonary artery would accomplish any good, and it might make the condition worse. This point, however, has not been determined. A significant reduction in the size of the main pulmonary artery combined with sympathectomy might result in improvement. Unfortunately, an entirely satisfactory method for the partial closure of large diseased arteries has not been developed, and there is always the danger of subsequent hemorrhage at the site if a sizable reduction in caliber is caused. All in all, the problem of the surgical treatment of the Eisenmenger complex and of high ventricular septal defects in general is a most difficult one. Its solution probably will have to await further developments in intracardiac surgery.

23. Pulmonary Hypertension in Association with Congenital Heart Disease: Pulmonary hypertension in association with a number of the congenital cardiovascular defects has already been discussed briefly. The question arises whether the alterations in the pulmonary arterioles are primary[63] or whether they are secondary to the congenital defect in the heart or great blood vessels. The future role of surgical therapy in the treatment of pulmonary hypertension in association with congenital heart disease is bound up so closely with this unsettled question of etiology that no dogmatic statements seem warranted. Even if it be granted that the pulmonary hypertension in the majority of cases is due directly to the alteration in circulatory dynamics caused by the cardiovascular defect, there is still the problem whether these changes in the pulmonary vessels will be reversed if the defect is corrected. These alterations in the pulmonary vessels are usually anatomic rather than vasospastic. It has been stated previously that in many cases the increase in the resistance in the pulmonary vascular bed serves as a safety mechanism in preventing an unbalancing of the systemic and pulmonary vascular systems. This is probably the case in the Eisenmenger complex, transposition of the great vessels, isolated ventricular septal defects, aortic atresia with patent ductus, the single ventricle in which both great arteries or the aorta alone arises from the rudimentary outflow chamber, and in some instances of truncus arteriosus. Even if the nature of the pulmonary vascular resistance were such that it could be lowered by sympathectomy without correction of the defect, severe circulatory disturbances would likely ensue. I am of the opinion that the pulmonary vascular changes are usually secondary to the cardiovascular defect and that the treatment should consist of a direct attack on the defect when and if a method is available. Naturally the best results will be obtained in those patients in whom vascular alterations in the lungs are not far advanced. If the primary defect can be remedied, it is possible that an additional procedure such as pulmonary sympathectomy will be helpful in reducing the pulmonary resistance, but there is no convincing evidence that the pulmonary vessels are under autonomic control. Unfortunately, surgical means are as yet unavailable for dealing with many of these intracardiac defects, and therapy will have to await further advances.

Discussion

Many significant advances in cardiovascular surgery can be and will be made by the employment of existing methods and techniques, but many others will have to await the further development of methods for carrying on the circulation artificially in order that one may operate under direct vision in a bloodless field. Still others are dependent upon the solution of the problem of homotransplantation of tissues. This latter problem is more baffling than the former, and no real strides have been made. It is to be hoped that some agent will be found that will so alter the donor-recipient relationships that transplanted tissues will survive and function permanently. The work with cortisone and ACTH along these lines is not very encouraging.

Important advances have been and are being made in studies on methods for carrying on part or all of the circulation artificially. Many of the needs for and future applications of these methods have been mentioned in this paper in the discussion of individual lesions.

Kolff and others who have worked on the artificial kidney have demonstrated conclusively that it is possible to withdraw blood continuously, to pass it through an artificial circuit, and to return it to the body without harm to the patient. The blood is removed from an artery of the patient, who has received heparin, is passed through the artificial kidney, and is returned to one of the patient's veins. The problem is more complicated if one wishes to take over the cardiorespiratory functions because the use of the artificial lung presents greater complexities than does that of the artificial kidney. In order to take over the cardiorespiratory functions, it is necessary to heparinize the patient, to withdraw blood from a vein, to expose it to oxygen and allow carbon dioxide to escape, and then to pump the oxygenated blood into a peripheral artery. As a result of the work of Gibbon and many others, oxygenators that function with remarkable efficiency have been constructed.

PARTIAL CIRCULATION

The mechanical lung and pump as partial replacement for the functions of the heart and lungs should be helpful to patients having difficulty in oxygenating adequately the blood which is to enter the systemic arterial system. Among other conditions, the procedure should prove helpful in the treatment of patients with severe pulmonary edema which cannot be relieved by conventional methods of treatment.

The remaining remarks on partial circulation will deal only with replacement of the function of the right or the left side of the heart, the subject's own lungs being used for the oxygenation of the blood. If and when the artificial lung is perfected and proved to be safe, it is likely that the use of methods for replacement of the function of one of the two sides of the heart will be limited to experimental work, since the cannulation of the great blood vessels is in itself an extensive and a dangerous procedure. Furthermore, there are many intracardiac and extracardiac procedures in which it is desirable to have both sides of the heart bloodless.

Leeds, Gray, and Cook,[69] Sewell and Glenn,[70] and others have rendered the *right side* of the heart bloodless, except for coronary venous return, by diverting the blood from the superior and inferior venae cavae to a pump and then through a cannula to the pulmonary artery. As indicated previously, this required the insertion of at least two large cannulae. Since the coronary blood flow equals 5 per cent or more of the cardiac output, difficulties are presented in operations on the right side of the heart from the viewpoints of visibility and blood loss. If the outflow from the coronary sinus and thebesian vessels is to be collected and returned to the circulation, it should be debubbled. Sufficient experimental evidence has been accumulated to demonstrate that a unilateral pump can be opened and closed with recovery of the animal.

A unilateral pump to replace the function of the right side of the heart temporarily should theoretically allow one to operate on lesions of the tricuspid and pulmonary valves, including stenosis, atresia, and insufficiency, on Ebstein's disease, on the rare infundibular stenosis unaccompanied by a septal defect, and on some intracardiac tumors. The unilateral pump would not suffice for the closure of septal defects or for operations for other lesions associated with septal defects. The pump will probably find its greatest usefulness in permitting one to create various experimental lesions of structures of the right side of the heart.

Zollinger,[71] Kantrowitz,[72] and others have used a unilateral pump to substitute for the *left side* of the heart. The blood from the left auricle or the pulmonary veins is withdrawn through a cannula and it is then pumped through another cannula into a systemic artery such as the subclavian. It is more difficult to substitute for the left side of the heart than the right. One of the reasons for this is the likelihood of arterial air embolism associated with opening the left side of the heart. Furthermore, the coronary arterial flow is dependent, when the left heart pump is used, upon the competence of the aortic valve. Nevertheless, Kantrowitz has reported survival of cats following opening and closing of the bloodless left ventricle.

A unilateral pump to replace temporarily the function of the left side of the heart should theoretically allow one to operate on lesions of the mitral valve and on intracardiac tumors. The coronary arteries would probably have to be cannulated and perfused if one were operating for aortic stenosis or insufficiency. The unilateral pump would not suffice for the treatment of septal defects. The present results on the treatment of mitral stenosis are so encouraging that the employment of a pump in this connection seems unlikely. Because of the dangers accompanying the use of the left heart substitute, and because of its limited clinical usefulness as visualized at present, it would appear that this method will be employed mainly in the creation of experimental lesions.

TOTAL CIRCULATION

Wesolowski[73] and others have shown that *both sides* of the heart may be rendered relatively bloodless by using two pumps, one for the right and another for the left. The connections made would be similar to those described under partial circulation; namely, one apparatus to withdraw blood from the venae cavae and propel it to the pulmonary arterial circulation, and another to withdraw blood from the left auricle or pulmonary veins and pump it to a systemic artery. Under these conditions the entire heart is deprived of blood except for the coronary flow, and various intracardiac procedures would be possible. It seems doubtful, however, whether the use of two pumps to replace the heart's functions will ever be a practical procedure in patients because the connections necessary between the two pumps and the patient make it too formidable. The method should continue to serve a useful purpose in experimental work.

Another method by which the function of the heart and lungs can be supplanted is by *cross circulation* between the recipient and a donor. This can be accomplished by having the donor's heart serving as the pumping mechanism, or mechanical pumps can be used. In either case, the donor's lungs would oxygenate the blood. Experiments indicate that the dangers to the donor are about as great as those to the recipient, and it would appear that this method will not have clinical application.

The last and best method is that in which an *artificial oxygenator* is used. The method involves cannulation of the venae cavae and passing of the venous blood through an oxygenator or artificial lung. The blood which has taken up oxygen and has lost carbon dioxide is then pumped to a systemic artery. Both sides of the heart are rendered relatively bloodless (if the aortic valve is competent) and intracardiac procedures such as operations on heart valves, septal defects, and intracardiac tumors are possible. The cannulation is simple as compared to that which is required when an oxygenator is not used. Those who are working in this field have had many problems to solve such as the prevention of damage to the blood and the prevention of air embolism. The two major problems remaining are the construction of a simple suitable oxygenator and the solving of the difficulties and dangers of operating on heparinized patients. Under the leadership of investigators such as Gibbon, Crafoord, Bjork, Kolff, Dennis, Glenn, Jongblood, and others, it is very likely that these problems will be solved. Only when this time arrives will it be possible to determine how well the diseased human heart will withstand having its functions taken over temporarily and incisions made into its chambers. It is probably fortunate that the dog's heart, because of its great tendency to fibrillate, presents a rigid test for the artificial circulation; the human heart presents less of a problem in this respect. It would seem that the initial clinical employment of the artificial heart and lungs for total replacement should be on patients with a usually fatal disorder such as congenital transposition of the aorta and pulmonary artery. The hearts of these patients withstand operative procedures remarkably well, and the use of the artificial circulation should give one an opportunity to divide and transpose the aorta and the pulmonary artery.

I hope it is obvious from my remarks that there are many unsolved problems in cardiovascular surgery. Many of these will be solved by modifications in existing techniques. Others will have to await further fundamental developments. With the interest and enthusiasm that are being shown by competent investigators, the future development of this field of surgery seems assured.

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EXPERIMENTAL RECONSTRUCTION OF TRACHEAL AND BRONCHIAL DEFECTS WITH STAINLESS STEEL WIRE MESH

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THE purpose of this report is to present the results of some experimental work done on animals involving the reconstruction of tracheal and bronchial defects with wire mesh.

With recent advances in thoracic surgery, more diseases of the lungs are becoming amenable to surgical therapy, and the limits of operability have been greatly extended. In the case of malignant disease, however, there are still several barriers to the width of excision. One of these barriers is the trachea. If malignancy involves the tracheal wall, it is either inoperable or, at best, resection can be only palliative. If some method of reconstruction or anastomosis of the trachea were possible, then a segment could be resected just as a segment of the intestinal tract may be resected. Such a method would undoubtedly extend the curability of pneumonectomy for bronchiogenic carcinoma and would aid in the treatment of primary neoplasia of the trachea. There are many other possible applications as well.

Experimental work in this direction has been carried out in the past, and is doubtless being undertaken by many at the present time. Definite progress has been made, and it does not seem at all unlikely that eventually a method or methods of reconstruction of the trachea and bronchi will be added to the armamentarium of the thoracic surgeon.

REVIEW OF PREVIOUSLY REPORTED EXPERIMENTS

Probably the first report on experimental repair of the trachea was made by Hohmeier,[1] of Munich, in 1911. He excised windows of cervical trachea in dogs, and repaired these defects with fascia lata. In one animal a defect of 1.5 by 1.5 centimeters was made, and in another 2.5 by 2 centimeters. Neither of these developed any emphysema, and at the end of eight weeks there was a completely regenerated smooth mucosa with slight stenosis.

In 1912, Levit[2] reported a tracheal repair on a human patient. He closed a small tracheal fistula, which had resulted in an attempt at suicide, by freshening the edges and suturing fascia over the defect. The patient was well eight months after operation, although there was slight narrowing of the trachea.

In 1917, Neuhof[3] reported the closure of an old tracheotomy which had failed to heal by suturing fascia into the defect. This patient was well six

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months after operation and had no apparent stenosis, although direct laryngoscopy was not done. He also mentioned several other methods of reconstruction which had been used. One was to turn down a flap consisting of superficial layers of the thyroid cartilage and adjacent skin, with the skin inside. Another was the use of an osteocutaneous flap from the sternum or clavicle. Nothing was stated as to the results of these procedures. He also mentioned that circular resection of the trachea had been done with success, but stated that it was difficult to get accurate approximation. In addition, he mentioned a case of tracheal fistula which was cured by implantation of a sheet of silver filigree. Unfortunately, no further details were given.

Taffel,[4] in 1940, reported a group of experiments on dogs in which he used free fascial grafts to close various defects. In the first group, 1 by 1 centimeter defects were made in the cervical trachea and repaired with free grafts of deep cervical fascia. All of these were airtight, and none of the animals had any respiratory complications or subcutaneous emphysema. In the second group, 5 by 5 millimeter defects were made in the left upper lobe bronchus, and were repaired with a free graft of fascia from the chest wall. Again there were no respiratory difficulties. In the third group a left upper lobectomy was done, and fascia was sutured over the open bronchus. And in a fourth group the tracheal defect resulting from pneumonectomy was repaired with a free fascial graft.

These procedures were done in twenty dogs, all of which healed primarily. There was no encroachment on the caliber except in the case of the lobectomy closure. The graft did not seem to remain viable, but rather acted as a scaffold for the regeneration of fibrous tissue. Regeneration of the mucosa was evident at the edges of the defect in one week and complete by the end of the second week, and there were mucous glands present at the end of the third week. In none of these was there evidence of regeneration of a muscularis mucosa, and, although there were islands of cartilage reformed, these did not take part in filling the defect. By the end of the fourth week, practically all healing had taken place. Microscopy revealed an inner layer of pseudostratified ciliated epithelium, a submucosa with occasional mucous glands and a few islands of cartilage but no muscularis mucosa, and an outer layer of supporting adult connective tissue.

It is true that these defects were quite small, but this report gave evidence that adequate functional regeneration of trachea and bronchi could take place.

Probably the most extensive study was reported by Daniel[3] in 1948. He first repeated Taffel's work, and in thirteen dogs made small 8 millimeter square windows. He then used three methods of repair. In several a fascial graft was used, in others the defect was closed with fibrin film, and in others he merely sutured muscle over the defect. In the first two groups he demonstrated regeneration of epithelium, although there was slight narrowing in the fascia group, and rather marked narrowing in the fibrin film group. In the third group, in which muscle was sutured over the defect, tissue protruded into the lumen, and there was some tracheal obstruction.

He then excised segments of thoracic trachea and tied the cut ends over glass, stainless steel, or vitallium tubes. This procedure was used in twenty-one dogs, of which six succumbed within five days. In the remaining fifteen he found that the tube was held firmly in place by fibrous tissue and granulation tissue, with one exception in which the tube had slipped down to the carina. In all cases the silk ligature, which had been used to tie the cut end around the tube, had cut through and was lying inside the lumen of the trachea against the tube. Islands of cartilage began to appear in three weeks and then grew slowly, and epithelium began to grow from the cut ends and was nearly complete in six months.

Daniel, in a third group, using eight animals, resected the right lung and the carina. He then tied the cut ends of the trachea and the left bronchus over a curved tube. All but two of these animals died of leakage, obstruction, or hemorrhage. The remaining two were sacrificed at six and fourteen months, respectively. In one, there was wheezing which gradually became worse. At six months there was almost complete regeneration of epithelium and, grossly, evidence of cartilage, but there was moderate constriction. In the other, the glass tube loosened and was removed bronchoscopically at eight months. There was slight narrowing noted at that time. At the end of fourteen months this dog had some wheezing, and at autopsy was found to have narrowing to one-half the diameter of the normal trachea. Epithelization was complete, and cartilage rings were present.

Hanlon[6] has recently reported on the use of gelatin sponge to repair several types of bronchial defects in dogs. He first demonstrated that, in vitro, gelatin sponge formed a relatively airtight barrier. In ten animals he made an incision in the anterior bronchial wall and wrapped gelatin sponge around the bronchus. This was successful in all cases. In the second group of ten dogs, small segments of the anterior bronchial wall were excised, up to 1 centimeter in width, and one-half the circumference. Again gelatin sponge was wrapped around the bronchus, using no sutures, but depending on its adhesive qualities alone. All but one of these animals survived. The one fatality was due to an infected pneumothorax on the third day. In a third group of fifteen dogs a lobectomy was done and the open end of the bronchus was closed by a cap of gelatin sponge, again without any sutures. Two animals died of empyema, but the remaining thirteen survived. After sacrificing the animals it was demonstrated that the repair would tolerate intrabronchial pressures much higher than those occurring normally. Microscopy demonstrated fairly rapid regeneration of epithelium, complete in several weeks. The gelatin sponge seemed to act as a scaffold for fibrous tissue regeneration, just as the fascial grafts had in previous experiments.

Since the completion of this present experimental work a very interesting group of cases has been published by Gebauer.[8] In cases of bronchostenosis he has used a graft of dermis laced with stainless steel wire to reconstruct the posterior bronchial wall. His technique consists, briefly, in incising the posterior bronchial wall at the stenotic area, allowing this to retract, then fashioning an oval-shaped graft of dermis which is laced with No. 28 wire to give it some rigidity. This graft is sutured into place with wire sutures to form a new, contoured, posterior bronchial wall. This method has been used in four clinical cases, all of which have been followed for a year or more. In all cases there seems to be satisfactory or fairly satisfactory function of the reconstructed segment.

PRESENT EXPERIMENTS

The reports of previous experimental work have demonstrated several things clearly. One is that regeneration of the trachea will occur. It seems that the regenerated wall is not complete in all elements, but does have a good mucosa capable of normal function. There is also a tendency to regeneration of cartilage, although it is not yet clear whether or not this regenerated cartilage will function well in maintaining a semi-rigid airway. Another fact demonstrated is one that might well be anticipated, and that is that there is a great tendency to some stenosis since the new wall is predominantly fibrous tissue, which tends to contract and thus diminish the lumen.

With these things in mind, the present experimental study was undertaken, using stainless steel wire mesh. This material was selected for several reasons. It has been demonstrated that stainless steel wire is quite inert and produces very little irritation. As a suture material it has been left in wounds for many years with no untoward effects. In the form of mesh, it has been used to repair large fascia-deficient hernias of the abdominal wall. Jonas[7] has demonstrated experimentally that the mesh becomes infiltrated and incorporated into the surrounding tissue with little or no inflammatory reaction. Fibroblasts seem to grow into the interstices and thus fix the mesh as an integral part of the new section of the abdominal wall. It is not completely rigid, but seems to have sufficient stability to prevent any recurrence of herniation. If this same type of healing were to take place in the trachea it would then be an ideal substance, since it would mimic the function of the tracheal cartilages and maintain a semi-rigid airway and prevent stenosis. Also, wire mesh may be moulded into a tube of any size, and thus the necessity of having a supply of various lengths and diameters of tubes would be eliminated. It would have an advantage over the rigid metallic or glass tube since the wire mesh could be used outside the tracheal wall and would not take up valuable intraluminal space, nor necessitate future removal with the possibility of subsequent stenosis.

FIRST GROUP

In this first group, mongrel dogs were anesthetized with intravenous nembutal and the cervical trachea exposed through a longitudinal incision. Two types of defects were then made. In some, one-half the circumference was resected, and in others the entire circumference (a segment) was resected. All the defects were made to extend over at least five tracheal cartilages. These defects were considerably larger than any of those previously reported, with the exception of Daniel's group of intrathoracic cases. Wire mesh (No. 40 or No. 80) was then wrapped around the entire trachea to form a tube outside the tracheal wall. No attempt was made to approximate the cut ends in the cases of segmental resections. Interrupted wire sutures were then used to suture the wire to itself, forming the tube, and to the trachea. The wire overlapped the edges of the defect by approximately two rings at each end, and in some cases the cut ends were sutured as well, in an attempt to prevent any retraction. The cervical fascia and muscles were then sutured over this repair and the skin closed.

No specific postoperative care was given, with the exception that a few were given systemic penicillin during a time when respiratory infections seemed to be quite prevalent among all the animals.

Some subcutaneous emphysema was anticipated since there had been no attempt at airtight closure, but surprisingly there was none.

SUMMARY OF RESULTS IN FIRST GROUP

There were ten animals included in this group with four deaths. One death was due to overdose of anesthesia, one from a massive wound infection on the sixth postoperative day, and two from pneumonia on the second and tenth postoperative days, respectively. It did not seem that any of these deaths were directly attributable to the type of operative procedure with the possible exception of the case of wound infection.

The remaining six dogs were sacrificed at periods varying from six weeks to five months. The one sacrificed at six weeks showed almost complete epithelization grossly. The two sacrificed at eight weeks showed grossly complete epithelization, and in one there was evidence of regeneration of cartilage. In one of these, one-half the circumference was resected and this trachea showed no loss of diameter and only slight evidence of scarring. In the other, a complete segment had been resected, and here there was very slight narrowing with a small area of granulation tissue on the anterior wall. However, there was an adequate lumen, and no evidence of respiratory obstruction. The dog sacrificed at four months, illustrated in Fig. 1, had an entire segment of trachea excised, and here there was complete epithelization except for some piling up of scar tissue anteriorly, which did not appreciably influence the size of the lumen. There was also regenerated cartilage (Fig. 2). The same results were found in the animals sacrificed at five months. Both of these had complete segments excised, and in both there was complete epithelization except for the piling up of scar tissue anteriorly. One of these is illustrated in Fig. 1.

In summary, epithelization appeared to be nearly complete in six weeks and complete in eight weeks. However, there was a tendency to an excess of scar tissue in the anterior portion of the wall in those dogs having entire segments resected. In only one of these was there any evidence of obstruction, and this was only wheezing. In two dogs the wire had been crushed with consequent diminution of the lumen. It is believed that this occurred from being picked up by their chain collars. This complication, although not too likely to occur, must definitely be kept in mind. Probably some remoulding could have been done endoscopically or by reoperation. In all the cases the wire mesh had been infiltrated by fibrous tissue, and appeared to maintain the newly formed wall as a semi-rigid tube.

It was difficult to obtain sections for microscopic study since the wire could not be sectioned and tissue could be obtained only by stripping it away from the

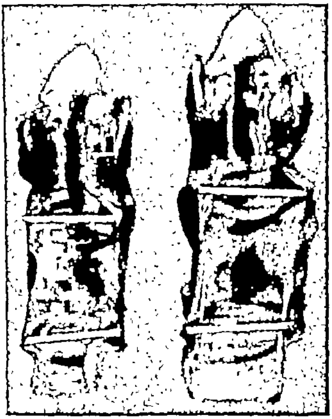

Fig. 1.—Left. Entire segment six rings (2.1 cm.) resected. Sacrificed at five months. Right. Entire segment five rings (2.3 cm.) resected. Sacrificed at four months. In both there is almost complete regeneration with re-epithelization except for slight piling up of granulation tissue anteriorly.

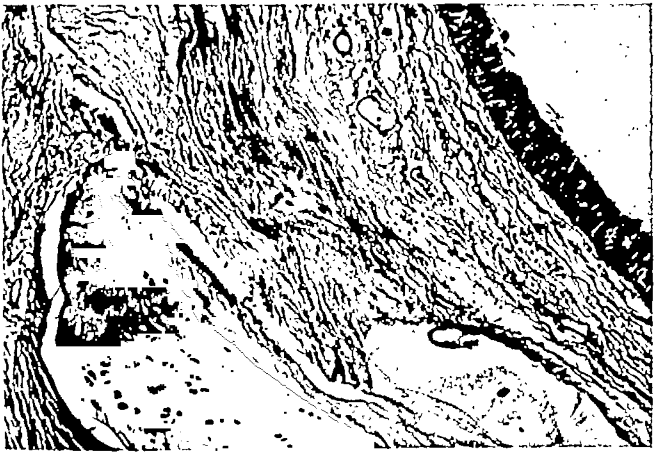

Fig. 2.—Photomicrograph of regenerated wall (200×) after four months.

mesh, to which it was quite adherent. Even so there was microscopic proof of normal respiratory epithelium in several. There was also both gross and microscopic evidence of regenerated cartilage.

SECOND GROUP

In the second group, mongrel dogs were anesthetized with intravenous nembutal, an endotracheal catheter was inserted, and artificial respiration given with intermittent positive pressure air. The right chest was then opened and the thoracic trachea exposed and freed, usually after sectioning the azygos vein. Similar segments of thoracic trachea were then excised, consisting of either one-half the circumference or the entire segment. Wire mesh was then wrapped around this defect with no attempt being made to approximate the cut ends. Again this mesh was sutured to the trachea and to itself with interrupted wire sutures. In the first case the parietal pleura was sutured over the reconstruction. This obviously did not form an airtight closure, and therefore several other methods of obtaining airtightness were tried. Free grafts of pericardium or skin were placed around the wire mesh and sutured to form an airtight covering. The chest was then closed and the pleural space aspirated. In most of these cases penicillin was given both by local instillation and systemically for a period of several days.

SUMMARY OF RESULTS IN SECOND GROUP

In this second group of eight animals, in which segments of thoracic trachea were excised and repaired, the results were rather disappointing since only one animal survived more than nine days. In the first animal, the repair was covered with pleura and this allowed leakage almost immediately with rapid demise of the animal. In the second animal, a graft of pericardium was used and this resulted in a good repair. However, in subsequent animals this could not be reduplicated. One animal died on the second postoperative day of pneumonia, with what was still an airtight repair. Another, was sacrificed on the sixth postoperative day since the wire mesh had not overlapped the cut ends of trachea sufficiently and had slipped, so that one edge of the mesh jutted into the tracheal lumen and caused obstruction. In this repair, however, there seemed to be no leakage. The remaining four deaths were due to leakage. In one, immediate leakage produced anoxia which led to cardiac arrest; in another, death occurred on the third day; in another, on the seventh day; and in the fourth, on the ninth day. The dog has a completely unstable mediastinum so that pneumothorax on one side results in a bilateral atelectasis and thus it seems that even the most minute leak will result fatally.

In one dog (see Fig. 3), a defect of four rings (1.5 centimeters), one-half the circumference was made. This was repaired with wire mesh covered by a free graft of pericardium. This dog recovered and remained completely well. After four and one-half months he was used for another experimental procedure, and at this time was intubated without difficulty. Following this he was sacrificed and it was found that there was absolutely no diminution of lumen. There appeared to be grossly complete epithelization and some regeneration of cartilage. This illustration seems to show some bare wire mesh, but this is only because of transillumination. The mesh was infiltrated by fibrous tissue and, although the wall was not so thick as the regenerated cervical tracheal wall, it was completely adequate. Microscopy did not show epithelium, but this is not surprising since the epithelium was stripped away from the mesh, to which it was quite adherent, and thus was damaged making adequate sectioning impossible. This result was considered satisfactory for all practical purposes, since it was completely airtight, the mucosa smooth, and the lumen normal in caliber.

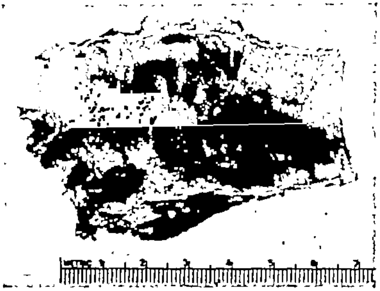

Fig. 3.—Defect of four rings, one-half the circumference of the thoracic trachea just superior to the carina. Sacrificed at four and one-half months. No narrowing and complete epithelization with beginning regeneration of cartilage. (Wire mesh is visible through intact epithelium.)

THIRD GROUP

In the third group, mongrel dogs were anesthetized with intravenous nembutal, an endotracheal catheter was inserted and artificial respiration given with intermittent positive pressure air. The right chest was then opened and a right pneumonectomy done. Either part or all of the carina was then resected so that a defect was formed which involved either the lateral half of the distal trachea and carina, or the entire distal trachea and carina. This was done to simulate a situation which is seen clinically. In some cases of carcinoma of the right bronchus there is extension to the carina or the lateral wall of the trachea. Such a lesion would be resectable if a method of anastomosing the trachea and left bronchus were available. These defects were then closed in a manner similar to that used in the second group, by making no attempt to approximate, but by bridging the defect with a tube of wire mesh. In this case the tube was tapered from the larger diameter of the trachea above to the smaller diameter of the left bronchus below. The wire mesh was found to be particularly suitable for this tapering. The mesh was sutured to the trachea and bronchus with interrupted wire, and then this area was wrapped with a full thickness skin graft which was sutured, or, in some cases, held by purse-string sutures at the ends and circular ligatures in the middle. Again in these cases the animals were given penicillin by local instillation and systemically for several days.

SUMMARY OF RESULTS IN THIRD GROUP

Again in this group the results were rather disappointing, since only one of the eight animals survived. One died of operative shock due to blood loss which occurred when the ligature on the pulmonary artery slipped. Two died of cardiac arrest, secondary to anoxia associated with leakage and obstruction, respectively. The remaining four deaths were due to leakage which occurred on the third or fourth postoperative day. Again, even a small leak would result in a pneumothorax with contralateral atelectasis.

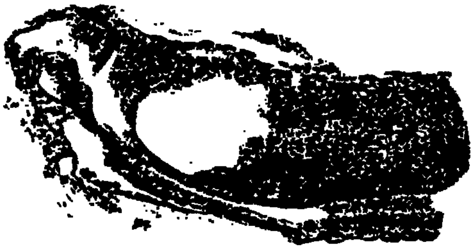

Fig. 4.

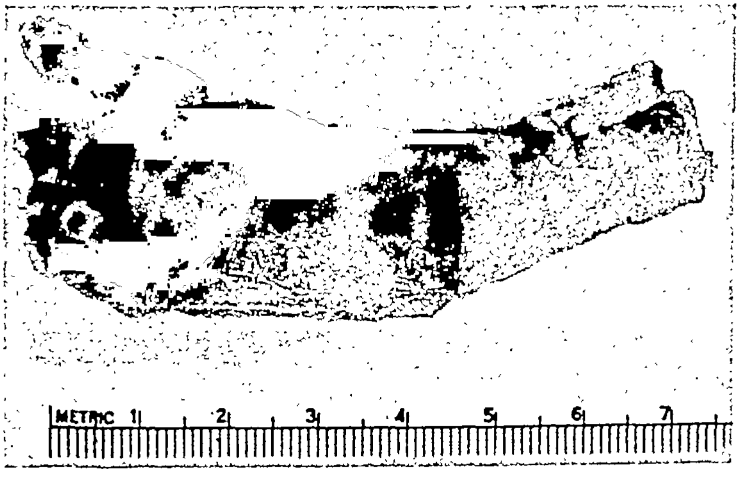

Fig. 5.

Figs. 4 and 5.—Right pneumonectomy with resection of one-half the carina and the right lateral half of the distal trachea to four rings proximal to the carina. Sacrificed at six months. Mucosal surface shows only beginning epithelization at edges with some bare wire mesh. External surface shows skin graft which apparently has been replaced by scar tissue. Repair completely airtight with no narrowing.

The survivor (see Figs. 4, 5, and 6) had a defect produced which included the lateral half of the circumference of the distal trachea and carina, extending to four rings above the carina. The wire mesh was placed and then covered with a full thickness skin graft which was sutured with interrupted wire. This animal remained completely well with no evidence of respiratory obstruction, and was sacrificed at six months. There was no diminution of the lumen, and the area of repair was quite clean. Regeneration had begun at the edges of the defect but was not complete, and a small window of bare wire mesh remained, which was airtight because of its having been infiltrated by fibrous tissue. The skin graft appeared to have become a thin sheet of scar tissue, but microscopy demonstrated some of the dermal appendages still intact, although there was no demonstrable epithelium in the section studied. Although healing was not complete, this was still considered to be an entirely satisfactory result since the reconstruction functioned well and there was no diminution of lumen.

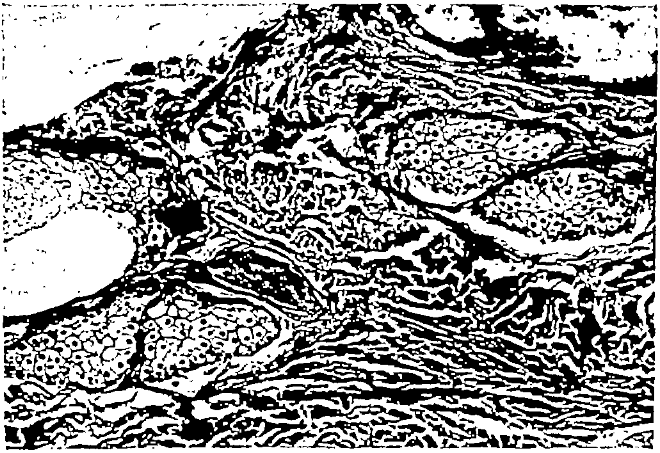

Fig. 6.—Photomicrograph of buried skin graft after six months. There is a fibrous stroma with scattering of chronic inflammatory cells, dilated structures resembling sweat glands, and a few sebaceous glands. No actual epithelium remains.

CLINICAL CASES

This method of repair has been used to a limited extent in two clinical cases which will be presented briefly.

Case 1.—A 42-year-old woman was admitted to the hospital with an undifferentiated carcinoma of the thyroid which invaded the anterior wall of the trachea. A thyroidectomy was done, and a portion of the anterior wall of the trachea was resected. This defect in the trachea was repaired with no. 40 wire mesh by merely suturing it over the defect, overlapping the cut edges by approximately 1 to 1.5 centimeters. The wire was not wrapped completely around the trachea as had been done in the experimental animals. The wound was closed by suturing the pretracheal muscles and skin. A tracheotomy was done below this point of reconstruction. The patient had an uneventful convalescence and had no evidence of tracheal obstruction, wearing a plug in the tracheotomy tube. In four weeks this area was examined by direct laryngoscopy, and there appeared to be complete regeneration of normal epithelium. There was only slight diminution in calibre due to slight flattening anteriorly. This patient subsequently was found to have a pulmonary metastasis, and after six months there was local recurrence in the neck, with eventual reinvolvement of the trachea and obstruction, necessitating the use of the tracheotomy. Unfortunately, no autopsy was obtained after the patient's death.

CASE 2.—A 38-year-old man was admitted to the hospital with a tracheal fistula. He had had a tracheotomy which had been closed on two occasions, only to break down and reopen. The trachea was exposed and no. 40 wire mesh sutured over the defect. The pretracheal muscles and fascia were then sutured over this. The patient had an uneventful recovery and remained well for eight months, although he failed to return for follow-up after that time.

CONCLUSIONS

This method of repair of tracheal and bronchial defects which has been described has been used on a number of experimental animals and on two clinical cases. The results in the cervical trachea are rather encouraging. There were no deaths directly attributable to the type of procedure used, and regeneration of the tracheal wall and mucosa seemed to be at least functionally satisfactory. The wire mesh became an integral part of the regenerated segment, and maintained it in good functional position as a semi-rigid tube. In some cases there was an overgrowth of fibrous tissue along the anterior wall which occasionally produced a minor degree of obstruction. This seemed to occur only in those cases in which an entire segment was resected, and it is quite possible that if this were used clinically such redundancy could be corrected by endoscopic cauterization. The crushing which occurred in two animals due to rough handling is something not likely to occur in human beings, and could still possibly be corrected endoscopically or by reoperation. In the two clinical cases, the method seemed to work well.

There are several possible clinical applications. In cases of malignancy involving the tracheal wall, as illustrated in one clinical case, the wall could be resected and reconstructed. Also, it may be of benefit in the closure of tracheal fistulas as demonstrated by the second clinical case. It may also find application in cases of tracheal stenosis, where the entire segment could be excised and then reconstructed.

The results of its use in the thoracic trachea seem rather disappointing, since there were only two survivors in this experimental group. However, nearly all of these were due to leakage and, since the dog has such an unstable mediastinum, even the most minute leak is fatal. It is possible that this would be handled in the human patient by continuous aspiration until sealing, just as is done following some segmental lobectomies or for bronchopleural fistula following lobectomy. In the two animals that survived, the result was entirely satisfactory.

Although this method of repair could certainly not be advocated for trial in the thoracic trachea of the human patient at this early date in its experimental development, still one might postulate some possible applications. The most obvious application would be its use in malignancy to enable one to do a wider resection and then reconstruct any trachea which had been sacrificed. It is conceivable that this might be applied to bronchial lesions, such as bronchial stenosis, either by resecting an entire segment or part of the circumference of the stenotic area and reconstructing an adequate conducting passage. Also there have been a few cases in which bronchi have been eroded by compression due to adjacent large lymph nodes. This wire reconstruction might be used merely to reinforce such an area of softening without actual resection.

In comparing this method to that of an intraluminal tube, the obvious advantage is that the wire mesh more closely approximates the function of the tracheal cartilages and is a part of the regenerated wall rather than a foreign body within the tracheal lumen. The disadvantages are just as obvious in that the wire mesh repair is undoubtedly more difficult and time-consuming than the ligation of cut ends over a solid tube. Also the difficulty in maintaining airtightness must be considered.

It may be that a combination of the two methods as suggested by Brown[9] would be more effective in the thoracic trachea. An intraluminal tube could be used to carry the patient over the time necessary for obtaining complete airtightness by fibrosis and then could be removed, leaving the wire mesh imbedded in the new wall and preventing later cicatricial contracture.

This paper is presented as a preliminary rather than a complete report. Additional work is being contemplated, and it is hoped that this present work will interest others to combine it with some of their studies, so that eventually there may be a relatively simple and satisfactory method of tracheal reconstruction to use in the human patient.

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The patients, both thoracoplasty and scapulectomy, were men ranging in age from 20 to 62 years, with the greatest concentration in the 20 to 29 year age group (37 per cent) and in the 40 to 49 year age group (34 per cent). The average age was 36.8 years.

Goniometric measurements were made on all patients studied, and all but three scapulectomy patients had roentgenograms of the scapulae. To the best of our knowledge all patients had normal shoulder girdle function preoperatively.

SCAPULOHUMERAL RHYTHM

It has been observed roentgenographically[3] that during the movement of abduction of the humerus both the scapula and humerus participate from the start. Except for individual variability during the first 30 degrees of abduction, the relationship between movement at the glenohumeral joint and the scapulothoracic joint remains fairly constant. For each 15 degrees of abduction of the humerus, 10 degrees of motion takes place at the glenohumeral joint and 5 degrees is contributed by the scapulothoracic joint, up to the full excursion of movement to 170 to 180 degrees. Throughout arm abduction the scapula remains in close contact with the chest wall and it rotates outward and upward, the inferior angle moving toward the axilla.

Most of the findings relating to the disturbance of scapulohumeral rhythm were gained from clinical observations.

NATURE OF SURGERY

The thoracoplasties performed at Castle Point entailed a five or six rib resection in two stages or a seven or eight rib resection in three stages, including the removal of the transverse processes. In most cases the posterior approach was used through the usual hockey stick type of incision. The shoulder girdle musculature traumatized during surgery consisted of the middle and lower fibers of the trapezius, the rhomboideus minor and major, the latissimus dorsi and the serratus anterior.

The partial scapulectomiees were performed either in conjunction with the last stage of the thoracoplasty or as a separate procedure (Table I). A variable portion of the distal scapula was resected in a manner to conform to the obliquity of the uppermost intact rib. The muscles which lose part of their scapular attachment are the serratus anterior, the rhomboideus major, the subscapularis, the infraspinatus and the teres major.

SHOULDER GIRDLE CHANGES FOLLOWING THORACOPLASTY

Skeletal Changes.—

After a thoracoplasty there occurs a variable degree of scoliosis, convex toward the operated side in the cervico-dorsal region, and often accompanied by some elevation of the shoulder girdle on the operated side (Fig. 2). However, the shoulder may be at the same level as the opposite side and infrequently it is lower (Fig. 1). Associated with a high shoulder one usually finds a deepening of the supraclavicular fossa.

Seen from a lateral view, an anterior displacement of the shoulder girdle on the operated side is often noted. The depression created by the resection of ribs permits the girdle to move forward into the decostalized area.

On the roentgenogram the scapula on the operated side is usually closer to the vertebral column than is its opposite (Figs. 1, 2). This is due in part to a medial displacement of the scapula and in part to the scoliosis with its convexity toward the operated side.

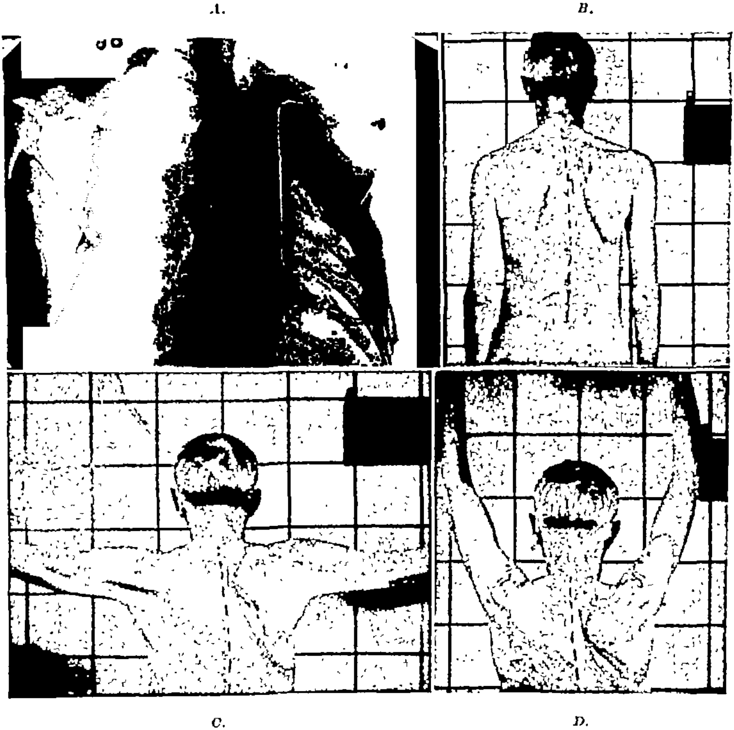


Fig. 1.—A 62-year-old patient who received a two-stage six-rib resection. Roentgenogram shows right shoulder girdle lower than its opposite, and scapula is closer to spinal column. Scapulohumeral angle on left is 37 degrees, on right 36 degrees. *B*, indicates some muscular atrophy in right supraspinous and interscapular areas. *C* and *D* reveal some disturbance in right scapulohumeral rhythm as evidenced by protruding vertebral border and superior angle of scapula, but arm abduction is about equal bilaterally: 156 degrees on left, 159 degrees on right.

Measurement of the scapulohumeral angle on the operated side of the ten representative thoracoplasty patients showed an insignificant difference from that of the nonoperated side. The difference ranged from minus 1 degree to plus 5 degrees, with an average deviation of plus 2.3 degrees.

A posterior view of the patient often reveals a groove on the operated side between the vertebral border of the scapula and the vertebral column, with some degree of prominence, posteriorly, of the vertebral border of the scapula (Figs. 1, 2). The groove is due to atrophy of the trapezius and of the rhomboids in the line of incision. When sufficient ribs have been removed to allow the whole scapula to sink into the decostalized area, a depression is commonly observed on the upper chest wall, especially in the thin patients, and the vertebral border of the scapula is no longer prominent. The heavy-set patients usually fill in the crests and crevices created by surgery, and their outer bodily contour is less disfigured.

Changes in Muscle Function.—

Experience has shown that, with the exceptions due to complications, the thoracoplasty patients gain normal (Fig. 1) or near normal range—within 15 to 20 degrees—of shoulder girdle movement as compared to the nonoperated side (Fig. 2).

The degree of loss of abduction of the ten representative thoracoplasty patients ranged from 0 to 18 degrees, with an average loss of 5.1 degrees.

Often the limitation of movement, when present, is due to soft tissue resistance, especially of the incised latissimus dorsi, which can usually be overcome with persistent effort.

A common finding following thoracoplasty is a slight disturbance of scapulohumeral rhythm associated with either normal or with some degree of limitation of shoulder girdle movement. This disturbance is evident during humeral abduction, at which time the vertebral border of the scapula is observed to protrude somewhat posteriorly from the chest wall (Figs. 1, 2).

SHOULDER GIRDLE CHANGES FOLLOWING THORACOPLASTY COMBINED WITH PARTIAL SCAPULECTOMY

Skeletal Changes.—

The partial scapulectomies performed at Castle Point consisted of the resection of a variable portion of the distal scapula, at a higher level medially than laterally. On the roentgenogram of the scapulae it was difficult to measure how much of the scapula had been excised because its vertebral border was often obscured by the regenerated ribs.

All of the roentgenograms made of the twenty scapulectomy patients have shown a medial and anterior displacement of the scapula on the chest wall (Figs. 3, 4, 5, 6, 7, 8, 9).

Sixteen of the 20 roentgenograms show some degree of elevation of the shoulder girdle on the operated side (Figs. 3, 8). An anterior view of the patient shows a deepening of the supraclavicular fossa accompanying the elevated girdle.

Seventeen of the twenty roentgenograms present a significant difference between the operated and nonoperated sides in measurements of the scapulohumeral angles, as compared to the roentgenograms of the thoracoplasty patients. The difference in the scapulohumeral angles of each scapulectomy pa-

Changes in Muscle Function.—

When abduction of the arms is initiated, a common finding on the operated side is a marked disturbance of scapulohumeral rhythm, characterized by a posterior protrusion of the vertebral border of the scapula with the scapula as a whole moving upward on the chest wall. As movement of both arms continues to a right angle, a lag of scapular rotation on the operated side is noticeable, more apparent in those with greater restriction of motion (Figs. 3, 4). When the patient is asked to stop the movement at shoulder height, although the

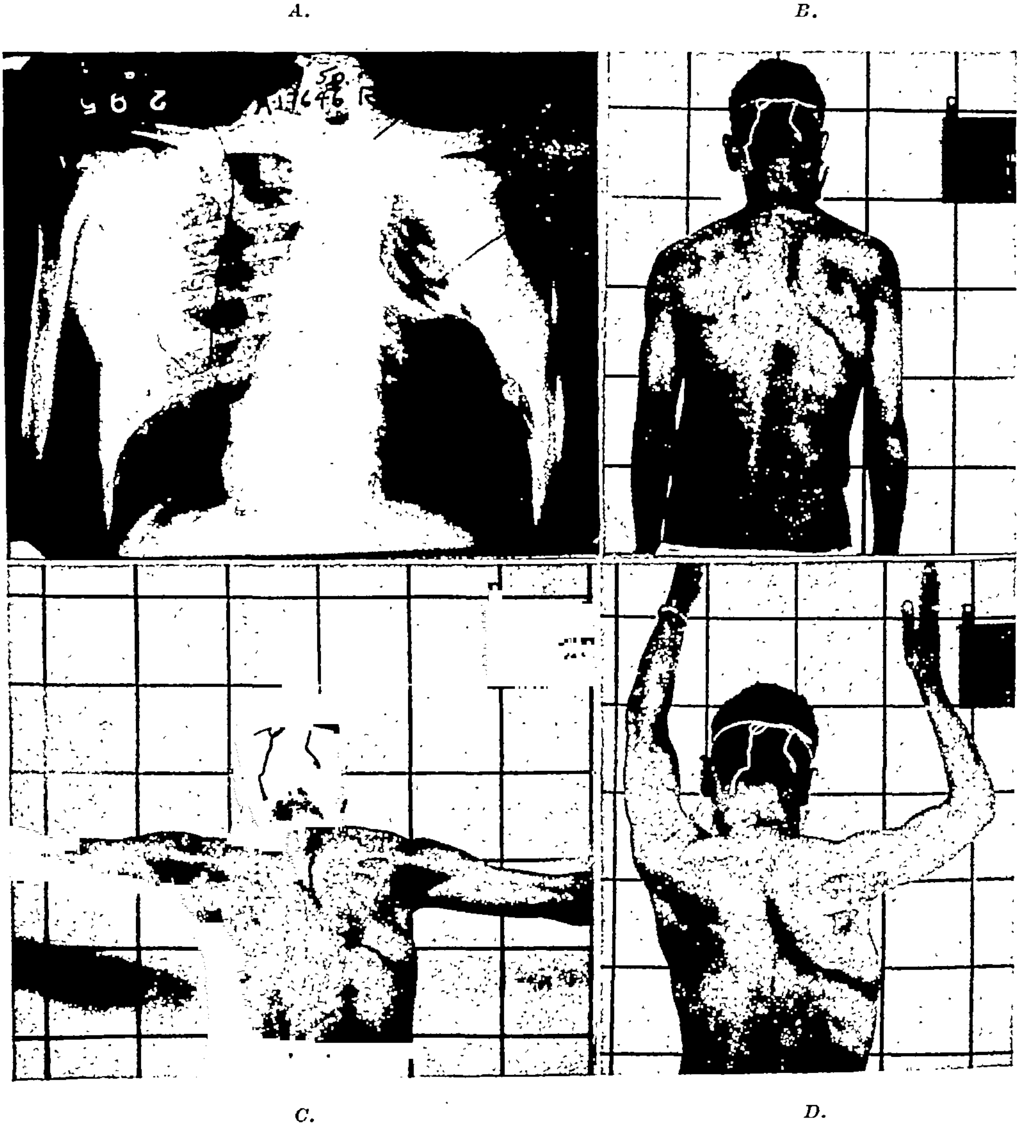


Fig. 3.—A 42-year-old patient with a two-stage, six-rib resection and a partial scapulectomy performed with the second stage. Roentgenogram shows right scapula displaced antermedially and rotated inward, with the superior angle elevated. Left scapulohumeral angle is 40 degrees, right is 58 degrees. Right upper chest wall in *B* presents a depression where scapula has moved anteriorly into decostalized area. With arms abducted to 90 degrees there is a lag in scapular rotation, right, and arm is at a lower level than its opposite. Also a prominence of right vertebral border of scapula may be noted. Range of full abduction in *D* is 170 degrees on left, 119 degrees on right. Right arm has moved somewhat anteriorly toward flexion.

operated scapula is higher than its opposite member, the arm as a whole is quite perceptibly at a lower level (Figs. 3, 4). Undoubtedly, when this occurs, the surgical damage to the scapular rotators is the cause; this will be discussed later.

As abduction continues beyond 90 degrees, in these patients, the arm of the operated side moves anteriorly toward flexion, the patient being unable to stabilize the shoulder girdle in the position of humeral abduction (Fig. 3).

When the scapulectomy patient has reached the maximum possible range of abduction, and if limitation of movement is present, the extremity can usually be moved passively to the full range but the patient is unable to maintain it there without assistance. The extremity tires easily in the elevated po-

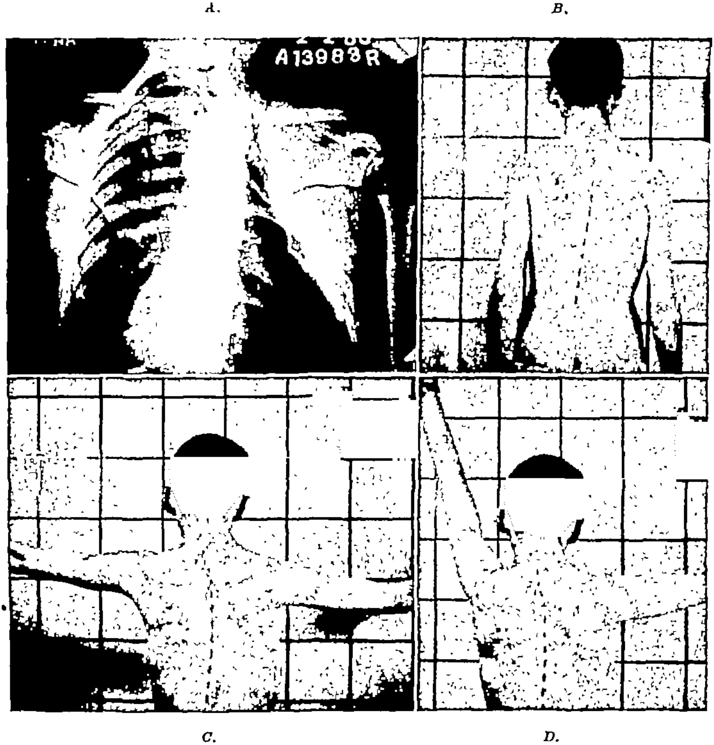


Fig. 4.—A 23-year-old patient who had five and one-half ribs resected in two stages, and a partial scapulectomy during the second stage. Roentgenogram shows right scapula displaced anteromedially and rotated inwardly, with the superior angle elevated. Left scapulohumeral angle measures 54 degrees, the right 33 degrees. *B* shows depression on right upper chest wall where scapula has been displaced anteriorly. At 90 degrees of arm abduction the operated shoulder girdle presents a prominence of the vertebral border and a lag in scapular rotation. Right scapula is elevated and the arm is at a lower level than its opposite. Arm abduction on left is 163 degrees, on right 100 degrees.

sition and must be brought down to rest. Even after six to eight weeks of daily physical therapy the attainment of active full range of shoulder girdle motion is not possible due to the limitation imposed by the surgical procedure.

Sixteen of the twenty-three scapulectomy patients showed a significant difference in the degree of abduction loss when compared to that found in the thoracoplasty group. The amount of restriction for the whole scapulectomy group ranged from 2 degrees to 85 degrees, with an average loss of 35.7 degrees. The thoracoplasty patients displayed an average loss of 5.1 degrees.

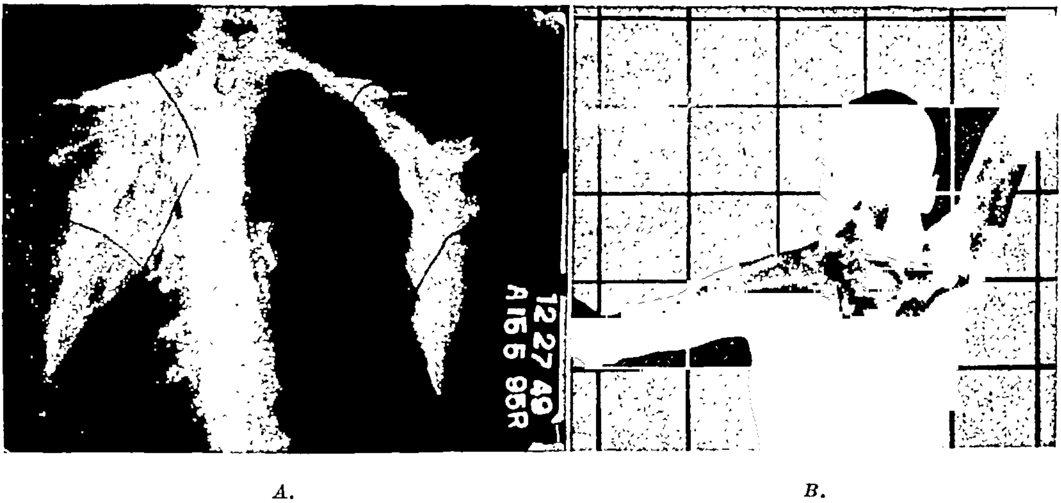


Fig. 5.—A 44-year-old patient who had a three-stage, seven-rib resection and a partial scapulectomy in conjunction with the last stage. Left scapula is displaced anteromedially, rotated inward, and the superior angle elevated, as seen on the roentgenogram. Scapulohumeral angle on left is 63 degrees, on right 42 degrees. In *B* the arms are abducted to their limit. Left scapula is elevated and the vertebral border protrudes posteriorly. Abduction on left is 72 degrees, on right 157 degrees.

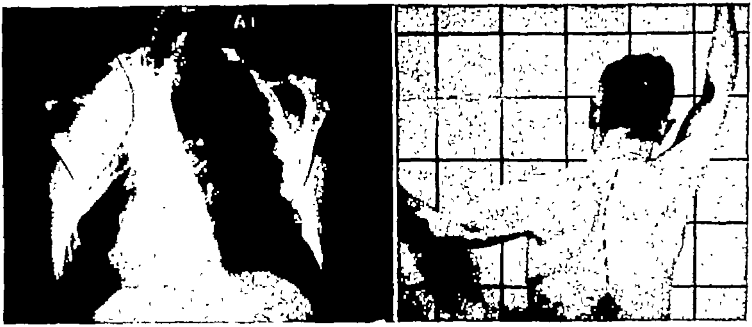
There appeared to be no consistent relationship between the scapulohumeral angle and the degree of abduction loss on the operated side. For instance, the patient with the greatest difference between his two scapulohumeral angles, that of 29 degrees, displayed an abduction loss of 63 degrees (Fig. 4). The patient with the greatest loss of abduction, 85 degrees, showed a 21 degree difference in scapulohumeral angles (Fig. 5). The patient with an 81 degree loss of abduction gave a scapulohumeral angle difference of only 11 degrees (Fig. 6). Still another patient with a 7 degree loss of abduction had a difference of 16 degrees in scapulohumeral angles (Fig. 7).

The question arises: "What are the factors that cause the increase in scapulohumeral angle and the degree of restriction of arm abduction?"

DISCUSSION

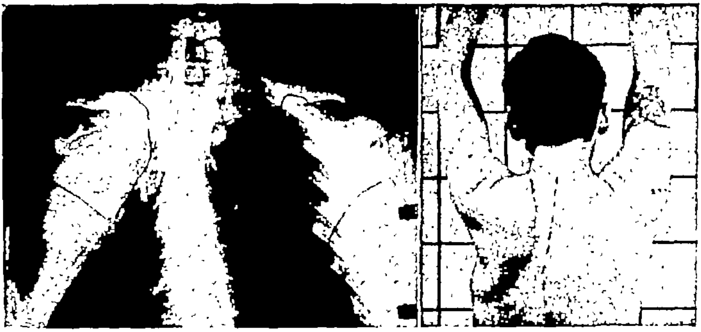
The disturbance of scapulohumeral rhythm observed on the scapulectomy patients is intimately related to the increase of the scapulohumeral angle and to the degree of restriction of humeral abduction. Let us examine closely the muscular dysfunction underlying these changes.

Scapular rotation during arm abduction normally elicits contraction from two groups of muscles, an upper and a lower group.[3] The upper component, consisting of the levator scapula, the upper fibers of the trapezius, and the upper portion of the serratus anterior, is not nearly as much disturbed during thoracoplasty and partial scapulectomy as is the lower component; i.e., the lower fibers of the trapezius and the lower digitations of the serratus anterior.



A. *B.*

Fig. 6.—A 58-year-old patient who underwent a three-stage, seven-rib resection and a partial scapulectomy with the last stage. Roentgenogram displays an anteromedial displacement and inward rotation of left scapula. Scapulohumeral angle on left is 45 degrees, on right 34 degrees. In *B* left scapula is elevated and the vertebral border protrudes posteriorly. Abduction is limited to 74 degrees on left, 155 degrees on right.



A. *B.*

Fig. 7.—A 23-year-old patient who had a two-stage, six-rib thoracoplasty and a partial scapulectomy with the second stage. Roentgenogram shows an anteromedial displacement and inward rotation of left scapula. Left scapulohumeral angle measures 63 degrees, the right 47 degrees. Range of arm abduction on left is 166 degrees, on right 173 degrees. Some prominence of left vertebral border of scapula is evident.

Trapezius and Serratus Anterior.—

The relationship between the trapezius and the serratus anterior in shoulder girdle movements is a most interesting one. Normally the two muscles act simultaneously to rotate the scapula during abduction of the humerus. When the trapezius is paralyzed, as in a spinal accessory nerve lesion, the serratus anterior alone is capable of rotating the scapula completely on the thorax and thereby permitting full range of abduction.[4-6] However, when the serratus anterior is completely involved, as in a long thoracic nerve lesion, the trapezius alone is incapable of rotating the scapula, and the humerus cannot be abducted much beyond a right angle.[4-8]

Normal scapulohumeral rhythm requires the coordinated effort of all the shoulder girdle muscles. However, if one may designate the relative importance of the various shoulder girdle muscles, the most important one, in the authors' estimation, is the serratus anterior. Without its function the shoulder girdle suffers greater disability than when any other single muscle is paralyzed.[6]

Scapulohumeral Angle.—

Particular attention is drawn to the function of the serratus anterior since this seems to be such a significant factor in the cause of the increased scapulohumeral angle and the limitation of abduction following partial scapulectomy.

Spalteholtz[9] notes that, of the three portions of the serratus anterior, the lowest portion, which includes the fourth to tenth digitations and converges to the inferior angle of the scapula, is the strongest.

Special cognizance is taken of the fact that the greatest concentration of the serratus anterior fibers occurs at the inferior angle of the scapula, and it is precisely this area that is excised during a partial scapulectomy.

Here is a good reason for the increase in the scapulohumeral angle after a partial scapulectomy. Normally, at rest, the serratus anterior maintains the scapula close to the chest wall and its vertebral border parallel to the spinal column. The loss of function of the lower fibers of the serratus anterior causes the scapula to rotate inward, the inferior portion moving closer to the vertebral column, with the angle between the humerus and the lateral border of the scapula (the scapulohumeral angle) thereby increased. On the roentgenogram the superior angle of the scapula is higher than its opposite (Figs. 3, 4, 5).

Probably the degree of inward rotation of the scapula is influenced by the amount of the distal scapula resected and by the manner of resection. The patient with the greatest increase of scapulohumeral angle appears to have had more than the usual amount of the distal scapula resected (Fig. 4). Those who showed a lesser increase of the scapulohumeral angle probably had less of the scapula resected. It was not always possible to distinguish on the roentgenogram how much was resected.

Of those scapulectomy patients who displayed no change or an insignificant change in the scapulohumeral angle, the manner of resection may have been modified. This was definitely the case in an operation observed by one of the writers (H. B. D.). The inferior angle and the medial and lateral borders adjacent to it were "trimmed" rather than resected in the usual manner. On the roentgenogram (Fig. 8) the scapula appears somewhat shorter than its opposite member but it retains something of its original configuration. In this instance, with the vertebral border relatively little disturbed and with less of the inferior angle removed, not only did the scapulohumeral angle show little change (3 degrees) but arm abduction showed a correspondingly negligible loss (5 degrees).

Degree of Abduction.—

Without scapular rotation the complete range of arm abduction is not possible. Scapular rotation and arm abduction will be interfered with to the extent that the lower fibers of the serratus anterior lose their area of insertion and with it their effective leverage on the scapula. Furthermore, the lower

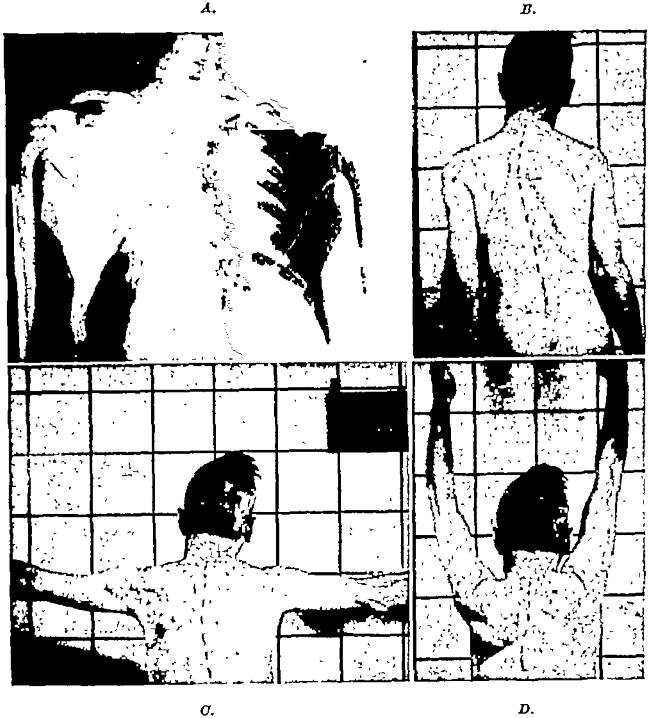


Fig. 8.—A 45-year-old patient who had a three-stage, seven-rib thoracoplasty at another institution, and a partial scapulectomy at Castle Point. Roentgenogram reveals an antero-medial displacement and elevation of left scapula. Scapulohumeral angle on left is 36 degrees, on right 33 degrees. *B* shows depression in left upper chest wall where scapula has moved anteriorly into decostalized area. In *C* the scapulae and arms appear to be almost symmetrical in position, with a slight disturbance of scapulohumeral rhythm (left vertebral border of scapula protrudes posteriorly). Full arm abduction gives a reading of 161 degrees on left and 166 degrees on right. Difference in range of abduction appears to be greater than actually due to deviation of head and neck to right.

fibers of the trapezius are weakened by the thoracoplasty and this in turn makes its contribution to the shoulder girdle dysfunction.

Involvement of the serratus anterior and of the lower trapezius manifests itself during arm abduction as a disturbance of scapulohumeral rhythm, the scapula moving upward on the chest wall and its vertebral border protruding posteriorly.

It has already been mentioned that there was no consistent relationship between the increase of scapulohumeral angle and the degree of abduction limitation. Occasionally some cause for diminished abduction may lie in the personality of the patient. Such a personality may be described, characteristically, as tense, apprehensive, and intolerant of pain.

The concept of the mechanism at work in this type of patient is that the surgical trauma, induced on a musculature already hypertonic, tends to increase the existing muscular tension. In addition, the low tolerance for pain discourages the patient from moving the extremity beyond the point of pain, however slight it may be. He tends to protect and favor the operated side. When the arm is moved it is done so reluctantly, the apprehension serving to increase reflexly the tone of the muscles antagonistic to those of the particular movement desired. When it is abduction of the humerus that is desired, the adductors of the humerus—the pectoralis major and the latissimus dorsi—increase in tension.

If this sort of reaction persists for any length of time one may well expect adhesion formation between the scapula and the adjacent tissues on the thoracic wall. Once this occurs the restriction in movement is marked and difficult to overcome. The patients in Figs. 5 and 6 illustrate just this condition.

Other Factors Affecting Shoulder Girdle Function.—

It should be noted as well that trauma to the various shoulder girdle muscles, other than those already discussed, contributes to shoulder girdle dysfunction. When the scapulectomy patients reach their individual limits of abduction, the affected arm often tires easily in that position. This may be due partially to the weakening or loss of action of the teres major in its capacity of assisting the arm to maintain a static position.[3]

Quite often it is observed that during abduction the arm advances toward flexion as it is elevated beyond the right angle (Fig. 3). Interference with the function of the serratus anterior which normally maintains the scapula close to the chest wall and of the rhomboids and the middle fibers of the trapezius which ordinarily fix the scapula in their plane of action prevents proper fixation of the scapula, thereby permitting forward movement on the chest wall during humeral abduction.

The subscapularis and the infraspinatus muscles are somewhat affected by a partial scapulectomy and this, too, probably weakens movement at the glenohumeral joint.

A previous study of thoracoplasty patients[1] indicated that age is not a significant factor in relation to the degree of skeletal deformity or in the changes of shoulder girdle function. Similarly, no relationship between age of patient and shoulder girdle changes could be found in the comparatively small number of patients included in this study.

The muscular development of the patient before surgery is a more important factor. This is best illustrated by a patient (Fig. 9) who was originally listed as having had a thoracoplasty only. Upon examination the shoulder girdle on the operated side behaved like that of a thoracoplasty patient with normal range of movement. However, the roentgenogram of the scapulae showed a difference of 17 degrees between the two scapulohumeral angles, which is a greater difference than that found within the range of the representative thoracoplasty patients. The roentgenogram further revealed a difference in the outlines of the

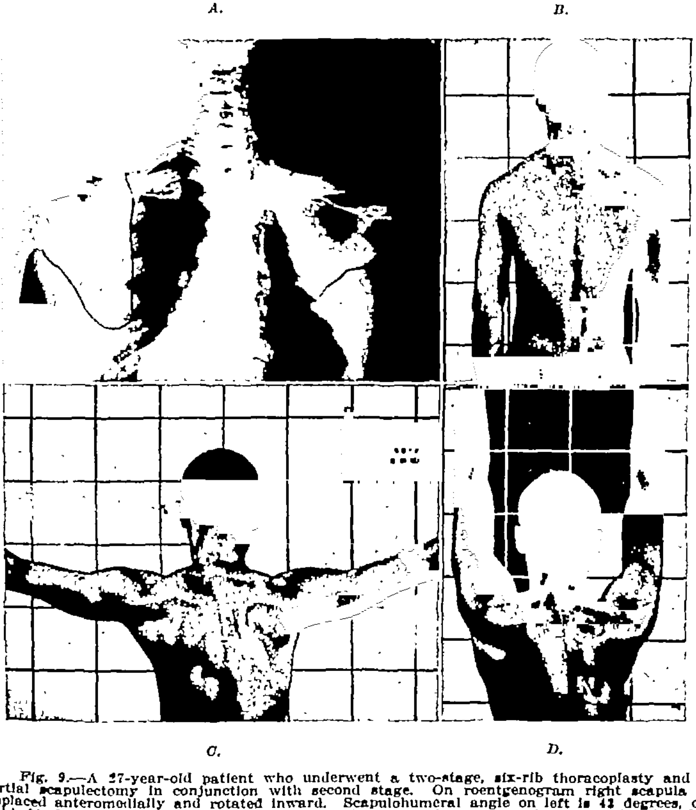


Fig. 9.—A 27-year-old patient who underwent a two-stage, six-rib thoracoplasty and a partial scapulectomy in conjunction with second stage. On roentgenogram right scapula is displaced anteromedially and rotated inward. Scapulohumeral angle on left is 43 degrees, on right 60 degrees. Depression in right upper chest wall is apparent in *B*. Right shoulder girdle is elevated slightly. Abduction of arms in *C* and *D* shows them to be equal and symmetrical, with a slight protrusion of vertebral border of right scapula. Abduction on left is 178 degrees, on right 177 degrees.

PULMONARY DECORTICATION FOR CLOTTED HEMOTHORAX FOLLOWING IDIOPATHIC SPONTANEOUS HEMOPNEUMOTHORAX

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THE treatment of clotted hemothorax by pulmonary decortication has become an accepted and often practiced procedure since World War II. Williams[3] reports a case of spontaneous pneumothorax with hemorrhage treated by decortication in 1947, and mentions a similar case reported by Elrod and Murphy[1] in which the patient was operated upon also in 1947. These latter authors[1] believe that their case is the first of this type to have been subjected to decortication. In a presentation on Feb. 18, 1949, before the New York Society for Thoracic Surgery, one of us (GPS) briefly mentioned a case of a patient with spontaneous hemopneumothorax treated by pulmonary decortication by Dr. Harold Neuhof in 1936, and also included a similar case of a patient operated upon by Dr. A. S. W. Touroff on Feb. 17, 1946. In order to attempt to establish priority for the procedure of decortication in the treatment of clotted hemothorax following spontaneous hemopneumothorax, a detailed report of the first case is indicated.

History.—R. T., No. 393576. A 23-year-old white man was admitted to The Mount Sinai Hospital on May 23, 1936, for the treatment of a collapsed right lung. Five and one-half weeks prior to admission, while at work as a clerk, he experienced sudden severe needle-like pain commencing in the right chest anteriorly just lateral to the sternum above the nipple and radiating straight through posteriorly. Pain persisted, but there was no immediate cough, dyspnea, fever, or hemoptysis. The following day a physician was consulted because of persistent pain, slight dyspnea, and vomiting. A thoracentesis was performed, and blood was removed from the right hemithorax. The patient was told that his lung was collapsed. He was admitted to another institution and repeated thoracenteses revealed old blood and serum. Roentgenograms showed complete collapse of the right lung with air and fluid in the right chest. Dyspnea decreased, but he developed a nonproductive painless cough. Pain disappeared and did not recur.

Physical Examination.—Trachea appeared in the midline, entire right chest moved only slightly with respirations, ribs were retracted, diminished fremitus was present anteriorly, tympany was noted anteriorly with moderate diminution of breath sounds posteriorly except at the base where there was flatness and absent fremitus and breath sounds.

Laboratory Data.—Hemoglobin 78 per cent, white blood cells 12,400, polymorphonuclears 79, lymphocytes 16, monocytes 4, eosinophiles 1. Sedimentation rate one hour and twenty minutes (for 18 mm. drop).

Roentgenogram on May 25, 1936, showed fluid and air in the right chest with a fluid level at the fourth rib anteriorly, the heart and mediastinum were drawn to the right, and the right lung was collapsed.

Discussion.—Although the bleeding had stopped, it was decided to explore the chest by open thoracotomy in order to attempt to release the collapsed lung and restore it to normal function. Since this was without precedent a great deal of discussion followed, and many arguments were advanced in favor of the conservative method of treatment by aspiration and watchful waiting. A review of the literature at that time failed to reveal any similar attempts of treatment by open thoracotomy to release the bound down lung. In fact, many authors were of the opinion that blood in the pleural cavity did not clot, but remained liquid

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and therefore was always absorbed without the formation of clot, restricting membrane, and resultant collapsed lung. Other authors claimed that the blood was defibrinated by the churning action of the heart, and the resultant fibrin might result in pleurisy and adhesions. As a result of these conflicting ideas, treatment consisted of either bed rest or aspiration of blood and replacement by air. However, there were a number of autopsy reports showing collapsed lungs as a result of previous hemopneumothorax with membrane formation. We had had similar experiences in cases which had permanently collapsed lungs following hemorrhage into the pleural cavity. In fact, at the time there was a patient on the same ward who had developed a total cholesterothorax following a steering wheel injury to the chest many years prior to admission, and he required a complete Schede thoracoplasty following evacuation of the cholesterothorax. There had also been some experimental work in animals to show that blood clotted in the closed thorax even more rapidly than outside the chest. The argument was advanced, therefore, that in view of the fact that blood clots in the thorax, a restricting membrane can and does form in certain cases, that permanent crippling of the lung can be produced by this membrane, at least some attempt should be made to avoid this chain of events by early thoracotomy and removal of the restricting membrane.

Operation.—May 23, 1936.—*Anesthesia:* avertin per rectum followed by gas-oxygen-ether by inhalation. A posterolateral incision was made in the seventh right intercostal space extending into the anterior axilla. Old blood clot which was present chiefly in the costophrenic sinus was removed and the field inspected. The collapsed lung was completely enveloped by a brownish homogeneous membrane extending from the prevertebral region to the anterior mediastinal area. This membrane was removed, and the lobes completely released. When the lung was inflated, it appeared perfectly normal. No obvious source for the bleeding was visualized, but it was assumed that it came from the lung. Layer closure was performed without drainage. Complete inflation of the lung was maintained as the thorax was closed.

Course.—Postoperative course was entirely uneventful. Roentgenogram on May 27, 1936, one day postoperative, showed almost complete reexpansion of the lung. On June 4, 1936, there was complete reexpansion of the lung. Patient was discharged well on the eleventh postoperative day.

FOLLOW-UP examinations and roentgenograms over a period of four years showed a completely expanded normal lung and the patient remained symptom free.

SUMMARY AND CONCLUSIONS

1. A detailed case report of a successful pulmonary decortication for clotted hemothorax following spontaneous hemopneumothorax is herein presented.
2. Attention is directed to the fact that this procedure was performed almost fifteen years ago, and probably represents the first operation of this type for spontaneous clotted hemothorax.
3. The rationale for the procedure and pertinent discussion is included.

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POSTERIOR MEDIASTINAL TRACHEOTOMY

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CASE REPORT

TRACHEAL obstruction at a level so low that it cannot be relieved by the usual cervical tracheotomy presents a difficult therapeutic problem. This is particularly true when the obstruction results from compression of the trachea by an aneurysm. The introduction through a cervical incision of a tracheotomy tube sufficiently long to pass beyond the obstruction is almost certain to be followed by erosion of the aneurysm and its rupture into the trachea. Renewed interest in the treatment of such aneurysms by polythene wrapping or wiring has once more emphasized tracheal obstruction as a complication of aortic aneurysm.[1-3]

More than forty years ago the question of low tracheal obstruction was extensively discussed and several solutions to it were suggested. In 1907 Gluck[4] proposed posterior bronchotomy by an extrapleural approach through the posterior mediastinum as a means of relieving this type of tracheal obstruction. It is not apparent that he ever carried out the procedure.[5] Other investigators decried this approach and felt it violated fundamental principles of thoracic surgery.[6, 7] Instead it was believed the solution lay in the formation of a lung or bronchial fistula[6-9] which, we understand, was intended to mean (1) an incision into the pulmonary substance to such an extent that a bronchus or bronchi of sufficient caliber for retrograde respiration would be opened; (2) the amputation of a lobe or portion of a lobe sufficient to accomplish the same purpose; (3) the transpleural construction of a bronchial fistula by an attempt to suture skin flaps directly to an incision in the bronchial wall. Kuttner[10] in 1908 first attempted to produce a bronchial fistula in a patient with compression of the trachea from an aortic aneurysm. An upper lobe was resected and the upper lobe bronchus fixed in the wound. This patient died a few hours postoperatively. In 1922 Rethi[7] reported the formation of a lung fistula by means of a two-stage operation in a patient with tracheal obstruction from an aneurysm. The patient lived fourteen months, twelve days, and respiratory exchange through the fistula was adequate. It was not until 1929 that an attempt was made by Jirasek[11] to perform a posterior bronchotomy as originally advocated by Gluck. Unfortunately this patient died the day following operation.

Attempts to relieve respiratory obstruction by posterior bronchotomy or the construction of a lung or bronchial fistula have been uncommon and, except for Rethi's case, almost uniformly unsuccessful. It has been more

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common to attempt to relieve tracheal obstruction by decompressive mediastinotomy as was suggested by Gulcke,[12] and more recently re-emphasized by Abbott.[2] As Abbott points out, however, several weeks must elapse following decompression of the mediastinum before the full benefits of the operation are obtained. In our patient, a 48-year-old man with severe respiratory obstruction from tracheal compression by a large aortic aneurysm, the severity and steady progression of the respiratory obstruction and the peculiar deformation of the trachea by the posteriorly projecting aneurysm made it seem unlikely that mediastinal decompression would restore unobstructed respiration. Instead, we chose to attempt the introduction of a tube into the trachea below the level of obstruction through an extrapleural approach in the posterior mediastinum. The proposed procedure was a modification of that suggested by Gluck many years ago.

CASE REPORT

History: J. N., U. H. No. 679024, a 48-year-old white man, was admitted to the Medical Service of the University Hospital on April 4, 1950, complaining of progressive difficulty in breathing. For several years the patient had had frequent winter respiratory infections which had increased steadily in severity. During these episodes his cough was productive of greenish-yellow sputum, and he was slightly dyspneic.

In October, 1949, the patient had an exacerbation of this illness. His cough became productive of three cups of greenish, thick sputum each day. He had continued fever and grew progressively more dyspneic. Respirations were labored and accompanied by wheezing. The patient had been hospitalized elsewhere at this time for eight days, receiving oxygen, penicillin, and streptomycin. He was discharged slightly improved, but his symptoms continued. There had been a twenty-five pound weight loss. He denied venereal disease.

Physical Examination: Temperature was 100.2° F.; blood pressure, 140/58; pulse, 92; respiration, 20 (labored).

On examination the patient was acutely ill and seriously dyspneic. There was an obvious expiratory tracheal wheeze. Expirations were prolonged and forced. Râles were heard over each lower lung field. Retromanubrial dullness was increased. A tracheal tug was felt. On auscultation of the heart there was a probable aortic diastolic murmur heard over the aortic area and along the left border of the sternum. A systolic murmur was present in the aortic area. The pupils were small and fixed. There was clubbing of the fingers. The patient was producing about 40 to 180 Gm. of purulent sputum each day. He was markedly cyanotic during frequent coughing attacks.

Laboratory Data: Kahn, 4 plus; hemoglobin, 14.0 Gm.; red blood cells, 4.73 million; white blood cells, 16,800; urine, normal findings.

Roentgenograms (which are not sufficiently clear for reproduction): Examination of the chest showed an aneurysm of the ascending, transverse, and descending aorta with a posterior dilatation which pushed the trachea posteriorly and to the right, greatly compressing it. There was extensive calcification in the aneurysmal wall. Angiocardiograms confirmed these findings but did not establish definitely the points of origin of the great vessels with respect to the aneurysm. Changes present in the right base were suspected of being pneumonitis or bronchiectasis. There was no evidence of vertebral erosion.

Course: Tracheoscopy was performed May 1, 1950. The trachea was reduced to a narrow slit at a distance of 20 centimeters from the upper gums by a mass projecting into the left tracheal wall. No pulsations of the mass were noted. On expiration the tracheal lumen was closed almost completely. Large quantities of brownish-green purulent secretions were coughed up during the procedure. The patient was given penicillin intramuscularly 400,000 units twice daily, and penicillin aerosol three times daily. The amount of purulent sputum decreased. Streptomycin, 0.5 Gm. intramuscularly twice daily, was added later.

On May 9, 1950, the patient was transferred to the Thoracic Surgery Service when it became apparent that the symptoms of respiratory obstruction were increasing steadily in severity. On May 22, 1950, the right posterior mediastinum was explored and a posterior tracheotomy carried out. Following local anesthesia of the pharynx a small endotracheal tube was introduced past the major point of tracheal obstruction. The patient was then anesthetized with ether-oxygen in a closed system. Through a vertical incision paravertebral segments, six to eight centimeters in length, of the third, fourth, and fifth ribs were removed. Extrapleural dissection was carried out, and the trachea, which deviated posteriorly and to the right, was exposed. The markedly compressed trachea was partially imbedded in the aneurysm. A longitudinal incision was made in the posterior membranous wall of the trachea approximately four centimeters above the carina. A number of Portex endotracheal tubes had been readied for introduction into the trachea but none could be sufficiently angulated to keep it in place. The surgeon was finally obliged to use a right-angled 38 French Pezzer catheter after removing its tip three centimeters beyond its angle. This was introduced into the trachea with the open end pointing toward the main carina.

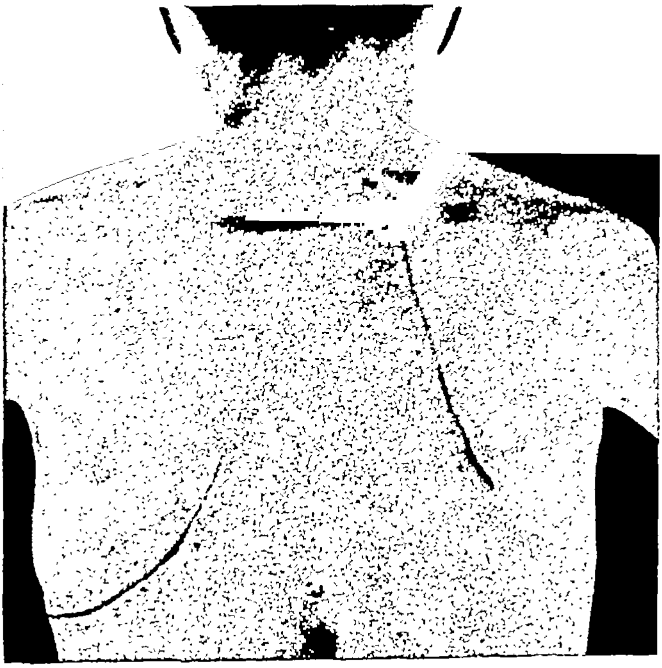

Fig. 1.—Photograph of the patient taken following insertion of the tracheotomy tube and subsequent left thoracotomy with partial wrapping of the aortic aneurysm.

The wound was closed about the catheter after it had been tacked to the prevertebral fascia and the fascia of the erector spinae muscles with a few interrupted sutures of 3-0 silk. A Penrose drain was placed down to the area of the tracheal incision. The small endotracheal tube that had been used during the operation was removed. Satisfactory respiratory exchange occurred through the tracheotomy tube. Following operation the patient was no longer dyspneic, and all respiratory distress disappeared.

Postoperatively there was no evidence of mediastinal infection or emphysema. It was possible to keep the transthoracic tracheotomy tube and bronchial tree free of secretion by means of a small catheter which could be passed beyond the angle of the tracheotomy tube. Constant "cold steam" was administered in conjunction with oxygen. After a few days 2 c.c. quantities of warm isotonic saline solution containing 80,000 units of penicillin were employed to irrigate the tube at from one- to three-hour intervals. The bronchial secretions, which had been so profuse immediately following operation, diminished progressively in amount and became less purulent in character.

On May 29, 1950, a left thoracotomy was carried out and the transverse and descending portions of the arch of the aorta were wrapped with strips of polythene film.* There was diffuse dilatation of the entire visible arch of the aorta. The descending aorta was also dilated. No definite involvement of the left carotid artery or left subclavian artery was present. *To the right of the vertebral column a secondary posteriorly projecting portion of* the aneurysm was found.

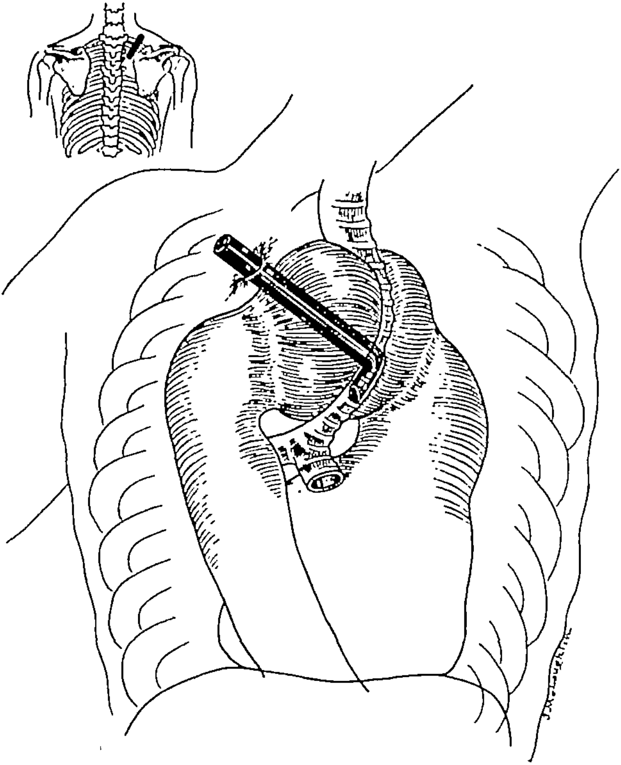

Fig. 2.—Diagram showing deformity of the trachea produced by the aortic aneurysm and the point of insertion of the tracheotomy tube.

The patient did well postoperatively on the same regimen, but on June 13, 1950, it was necessary to change the tracheotomy tube which had become plugged with dried secretions. A new tube, similar to the original, was reintroduced into the trachea through the back by threading it over a four millimeter bronchoscope and guiding the tube into the trachea under direct vision. Thereafter, no further obstruction of the tracheotomy tube occurred.

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*1.5 mil. solvent cast DuPont Polythene, NV-7-14, furnished through the courtesy of E.I. duPont de Nemours and Company, Wilmington, Delaware.

Subsequent to this, on July 3, 1950, and on July 7, 1950, attempts were made to remove the tracheotomy tube permanently, but it became apparent that it had been inserted very near to the point of greatest obstruction of the trachea and that the left-sided wrapping of the aneurysm had not as yet significantly decreased the aneurysm's pressure against the trachea.

On July 21, 1950, a right thoracotomy was performed and that portion of the aneurysm visible in the right chest was wrapped with polythene film. The flattened, compressed trachea, which had been pushed far to the right and posteriorly, lay partially imbedded in the thin, pulsating wall of the aneurysm. The ascending portion of the arch of the aorta was greatly dilated, and it expanded posteriorly as a secondary aneurysm which involved the trachea. The innominate artery did not appear to be involved. Dissection was carried medially to the limits of the previous wrapping. The trachea was completely freed from the aneurysm.

The patient again recovered satisfactorily. He maintained a weight gain of twenty-two pounds and had no respiratory distress. Suctioning was required infrequently, and the patient no longer needed ''cold steam.'' He became ambulatory and felt well for the first time in many months.

On Aug. 22, 1950, the posterior tracheotomy tube was permanently removed. The trachea was found on bronchoscopy to be widely patent, although there were granulations about the tracheotomy wound. The patient was able to breathe normally when the tube track was occluded.

On Sept. 1, 1950, bronchoscopy was again carried out. The healing tracheotomy wound was seen with difficulty. There was little evidence of tracheal compression by the aneurysm. The patient was discharged on Sept. 3, 1950.

On Sept. 27, 1950, the patient was readmitted complaining of weakness and increasing dysphagia. There were no respiratory symptoms. Bronchoscopy was performed on Oct. 2, 1950. This demonstrated some bulging inward of the left tracheal wall, but an adequate airway was present. During the evening of Oct. 2, 1950, the patient began to vomit bright blood and to pass tarry stools. Bleeding continued, and he died on Oct. 4, 1950. We assumed that the aneurysm had ruptured into the esophagus. Autopsy was not permitted.

DISCUSSION

The satisfactory maintenance of a posterior mediastinal tracheotomy for more than four months in a patient who had profuse, purulent, pulmonary secretions complicating severe tracheal obstruction has demonstrated the feasibility of respiration by this route. Obviously, careful nursing care was of the utmost importance in the result obtained. The right-angled catheter that was necessarily used for this patient was not an ideal tracheotomy tube for the purpose. Had it been possible to employ a tube with a more gradual curve, aspiration of secretions from the bronchial tree would have been facilitated. Despite the angulation of the catheter, however, the tube remained unobstructed except for one episode early in the patient's postoperative course when the nursing technique for the care of the tube was being evolved. Every effort was made to prevent secretions from drying in the tube. During the time when oxygen was necessary, it was administered with "cold steam." In addition, at one-, two-, or three-hour intervals the tracheotomy tube was irrigated with 2 c.c. of warm isotonic saline containing 80,000 units of penicillin. Local instillation of this solution, combined with the systemic use of penicillin and streptomycin, aided in progressively diminishing the quantity and purulent character of the bronchial secretions.

Although the tracheotomy tube had been in place more than four months, a few hours after it had been removed air could not be blown out of the sinus track and during subsequent bronchoscopic examinations it became difficult to visualize the tracheotomy site. In all probability the freeing of the embedded trachea from its site in the wall of the aneurysm contributed more to the restoration of an adequate tracheal lumen than the fibrosis following wrapping of the aneurysm with polythene film.

Mediastinal decompression carried out early in the patient's hospital stay might have been equally effective in relieving his tracheal obstruction. Certainly any attempt to produce a lung fistula or bronchial fistula would have been a much less satisfactory solution of this desperate problem.

SUMMARY

Posterior mediastinal tracheotomy was carried out in a patient with low tracheal obstruction from an aneurysm of the arch of the aorta. Respiration was maintained satisfactorily by this means for more than four months. During this period left and right thoracic explorations were performed in order to wrap the aneurysm with polythene film. The tracheotomy tube was removed and the patient breathed normally for one month. Death then occurred from hemorrhage following erosion of the aneurysm into the esophagus.

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A BULLET EMBOLUS TO THE LEFT FEMORAL ARTERY FOLLOWING A THORACIC GUNSHOT WOUND

Probable Entrance Through Thoracic Aorta

Case Report and Résumé of Peripheral Arterial Bullet Emboli

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THE popular and reasonable concept that a bullet wound of the heart or of the great vessels connected with it is rapidly fatal due to hemorrhage has a number of exceptions. The literature contains many interesting and intriguing reports concerning the mode of entrance and behavior of intracardiovascular foreign bodies. Those in which there was peripheral arterial embolization of the foreign body are summarized and such a case is herewith reported.

CASE REPORT

O. H., a Negro laborer aged 27 years, entered Cook County Hospital as an emergency case at 5 A.M. on Nov. 13, 1944, complaining of pain in his left groin and leg and inability to walk. While in a standing position half an hour earlier he had been shot twice, in the right arm and right flank, had immediately noticed pain in his left groin and soon thereafter pain throughout his entire left leg. He was able to take two or three steps before his left leg buckled under him and he fell to the floor.

On admission to the hospital, he was in fairly good general condition. His temperature was 99° F., pulse rate measured 96 beats per minute, respiratory rate 32 per minute. The blood pressure was 124/80. One bullet entered just anterior to the right greater trochanter. The other bullet coursed through the soft tissues posterior to the right humerus at its midpoint and entered the chest through the fifth interspace in the posterior axillary line. There was no Horner's syndrome. The pupils were round, equal, and reacted to light and accommodation. The heart was entirely normal. There was dullness at the base of the right lung posteriorly with diminished breath sounds. The abdomen was soft, and peristaltic sounds were present. The right leg was negative. The left leg was cold and paralyzed below the level of the knee. There was a pulsation in the left femoral artery just below the Poupart's ligament, but none in the popliteal artery nor in the posterior tibial or dorsalis pedis arteries.

Erythrocytes numbered 4,100,000, and the hemoglobin level measured 75 per cent normal. The Kahn test was later found to be negative. Roentgenograms showed no foreign body in the chest where one was expected, but the shadows of two bullets were present in the film of the pelvic region. The bullet

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which entered the right hip lay along the inferior ramus of the right pubic bone. The other bullet lay in the position occupied by the left femoral artery immediately over the head of the left femur at the point where the profunda artery arises (Fig. 1).

The nature of the disturbance in the left leg was not immediately realized, and it was thought that the bullet which entered the region of the right hip had damaged the lumbosacral plexus on the left, resulting in paralysis. A neurosurgical consultant felt that the findings could be accounted for entirely on the basis of vascular occlusion. Although approximately eighteen hours had elapsed since the accident, it was considered advisable to remove the bullet.

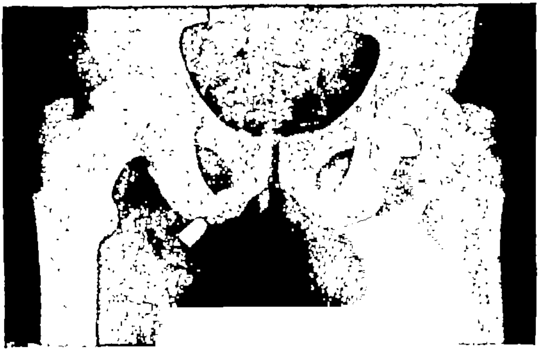

Fig. 1.—Roentgenogram showing positions of two bullets. The foreign body shadow overlying the head of the left femur represents a bullet in the femoral artery.

Under satisfactory local anesthesia the left femoral artery was exposed. The bullet was found in the artery just at the point where the profunda branch arises (Fig. 2). The bullet was extracted and a tail clot was removed. The artery was closed with a continuous mattress suture of silk everting the edges of the arteriotomy wound. The patient's general condition was satisfactory after the operation. Postoperatively no anticoagulant therapy was immediately available and none was used in the subsequent management. The patient was treated for a time by continuous caudal anesthesia, Novocain injections of the lumbar sympathetic trunk, together with papaverine and the use of venous occlusion in order to dilate the collateral circulation.

Approximately ten days after removal of the bullet, gangrene of the toes developed and gradually extended to involve the leg below the knee. Before a line of demarcation appeared considerable infection in the gangrenous leg occurred. The temperature ranged from 103° F. to 105° F., and the blood culture on one occasion was positive for staphylococcus aureus. Twenty-two days after the accident a supracondular amputation was performed. The wound healed by primary intention and the patient was dismissed from the hospital on the fortieth day.

Sixteen months after the accident the patient was in good general condition and was using an artificial leg satisfactorily. The blood pressure readings in both arms were normal and equal as were the pulses. Fluoroscopic examination of the chest revealed no dilatation of the aorta, no enlargement of the mediastinal shadows, and a clear retrocardiac space. A roentgenogram of the pelvis showed the bullet in place along the inferior ramus of the right pubic bone. Recent efforts to trace this patient have been unsuccessful.

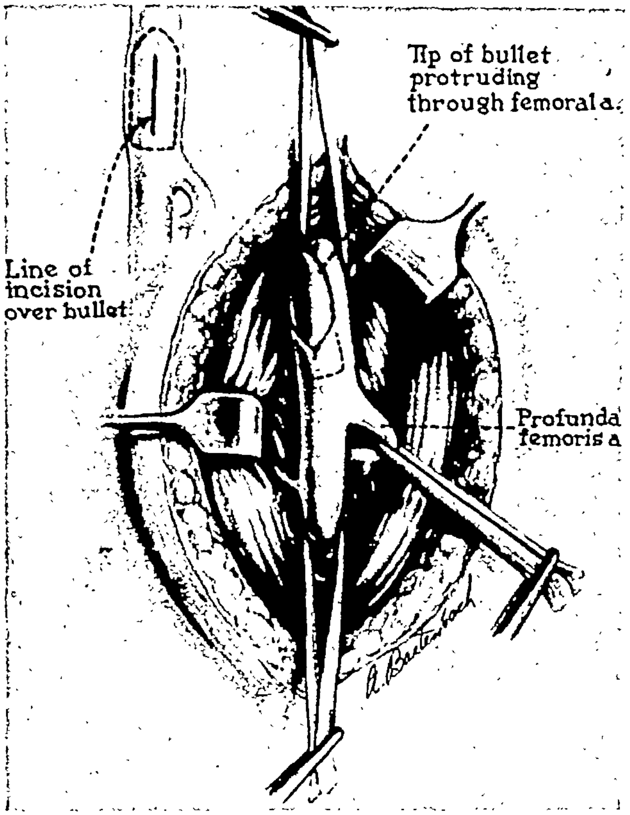
Tip of bullet protruding through femoral a.
Line of incision over bullet
Profunda femoris a

Fig. 2.—Sketch of the operative field showing position of bullet at time of embolectomy.

ABSTRACTS OF PREVIOUSLY REPORTED CASES

Twenty-two cases gleaned from the available literature are summarized. Additional details of the cases presented by Straus[1] appear to justify their inclusion in this report.

The following six patients survived bullet wounds of the chest with peripheral arterial embolization of the missile:

Case 1.—Schloffer,[2] 1903. A coachman, aged 18 years, shot himself in a suicide attempt while driving a carriage. He was admitted to the hospital two hours later, unconscious and in shock, with rapid and shallow respirations. The heart sounds were normal; a faint pulse was felt at the left wrist but none on the right. The wound of entrance was just to the left of the midline of the sternum at the level of the nipples. No wound of exit was found. The next morning the patient was conscious and complained of severe pain in his right arm, which was cyanotic, cooler than the left, and had no pulse. Motor and sensory paralysis was present below the level of the elbow. A foreign body, the bullet, was palpated high in the right axilla. Two days later there was fever. Dullness was present at the base of the right lung for about ten days. On the twenty-fourth day roentgenograms showed the bullet to be in the right axillary region.

At operation two months after injury a 7 millimeter bullet with an attached thrombus and "a few drops of pus" were found in the axillary artery at its beginning. The segment of the artery containing the bullet was removed and the ends ligated. There was some improvement in the arm but disturbed motility persisted. There was enough disturbance in sensation so that the patient unwittingly burned his fingers. Two years later he was examined by Prof. von Jaksch, the internist, who found the heart to be enlarged, and findings diagnostic of mitral stenosis and insufficiency were present. The hand and arm were unchanged.

CASE 2.—Rubesch,[3] 1912. A man, aged 28 years attempted suicide. The bullet entered between the third and fourth interspace 1.5 centimeters medial to the left mammary line. He remained standing, developed dyspnea, and when seen in the clinic one hour later had severe pain in the entire right leg, which was cooler than the left. Both femoral pulses were present. Roentgenograms revealed no bullet in the chest, but the shadow of the bullet was found just inferior to the head of the right femur, 5 to 6 cm. below the inguinal ligament. Embolectomy two days after the injury revealed a thrombus proximal and distal to the bullet, which lay transverse in the femoral artery. Discoloration of the foot began two days after embolectomy. Three amputations were performed; the first, fifteen days after the shooting, the last, above the knee, two months later. Two and one-half months after discharge from the hospital an x-ray of the chest was normal, but the patient had a systolic murmur at the apex of the heart. The bullet was thought to have gained entrance to the circulation by way of the heart.

CASE 3.—Boeckel,[4] 1917. An infantryman was shot in the right shoulder at the level of the deltoid insertion, and subsequently developed hemoptysis. On the third day roentgenograms showed the missile to be located in the left ventricle. The bullet embolized to the left femoral artery on the fourteenth day, at which time embolectomy was done. Gangrene of the leg developed, and it was amputated on the eighteenth day. The patient was dismissed from medical care on the thirty-fourth day. The missile was described as "a German bullet."

The case of Maurizot[5] so closely resembles that of Boeckel as to the missile, "a German bullet," the point of entrance, symptoms, the intervals before embolectomy, gangrene, amputation, and dismissal that the two authors seem to have described the same case independently.

CASE 4.—Perdoux' case, presented by Baumgartner,[6] 1919. A soldier, age not stated, was wounded by an airplane machine gun bullet which entered in the right posterior axillary line just lateral to the scapula. He was unconscious for a time but was in good general condition when examined nine days later. On the thirteenth day, roentgenograms showed the missile to be in the anterior part of the heart near the diaphragm. The point of the bullet was directed toward the base of the heart. Twenty days after injury roentgenograms and fluoroscopy showed the bullet in the same region with the point immobile, but the base oscillated with the cardiac movements. Thirty days after injury the missile was absent from the cardiac area, but was seen in the region of the right common iliac artery. The migration had not caused any symptoms. There were no pulses palpable in the right leg, and the blood pressure at the level of the calf was 50/30. On the forty-third day 1,200 cubic centimeters of serosanguineous fluid were removed from the right pleural cavity. Culture of it was negative and the temperature was normal. On the eighty-seventh day roentgenograms showed the bullet lying obliquely in the region of the right common iliac artery. At that time the patient had no dyspnea and walked easily but had cramps in his leg while climbing stairs. On Dec. 21, 1918, ninety days after the accident, laparotomy was performed, the right iliac artery was opened, and the bullet was removed. The artery was sutured, but the circulation was not restored. Pulsations did not return but the efficiency of the circulation improved. Six months after injury the patient could walk fast without discomfort, but noted weakness in the leg on climbing stairs. There were no cardiac symptoms and no abnormal cardiac findings.

The author thought that the early immobility of the bullet meant that the point was fixed in the anterior wall of the ventricle and the base in the posterior wall. The movements of the heart loosened the base and as it gradually entered the cavity of the vertical the wound in the posterior wall healed behind it. The base was then free to move in the cavity of the left ventricle. It was not until the movements of the heart freed the point of the bullet from the anterior wall of the ventricle that embolization occurred.

CASE 5.—LaRoque,[7] 1926. A male, aged 36 years, was shot from above while running downstairs. He continued to run "a short distance" when the left leg gave way; pain and weakness developed in it. Examination disclosed the bullet hole medial to the ventricle border of the left scapula, and a large effusion in the left thorax. Roentgenograms showed bullet fragments and fluid in the left chest, but the main part of the bullet was found by roentgenogram in the region of the left femoral artery. The patient was treated symptomatically and, on the eightieth day after the accident, 2 inches of the left femoral artery containing the bullet and a similar length of the accompanying vein were resected just above the profunda branch. Quadruple ligation of the vessels was done. The postoperative course was uneventful, and the patient was ambulatory in two weeks. Efforts to trace this patient after dismissal were unsuccessful.

CASE 6.—Cooper and co-workers,[8] 1948. A soldier, aged 22 years, sustained a penetrating wound of the left arm and chest from a 45 caliber bullet on May 26, 1945. He was admitted to a hospital shortly thereafter. The bullet penetrated the lateral aspect of the left arm, fractured the humerus, emerged on the medial aspect near the axilla, and then entered the chest between the fourth and fifth ribs in the axillary line. A hemothorax developed. The fracture of the humerus was treated and the wound of the chest was débrided and sutured. A swishing murmur was heard over the sternum on the fifteenth day. On the twenty-sixth day he suddenly complained of pain, coldness, and numbness in the right foot. The femoral pulse on the right disappeared the next day, and the pulsations in the left leg were diminished. Roentgenograms three days later showed the bullet at the bifurcation of the aorta. Seven months later, after two attacks of pain in the chest and abdomen, the segment of the abdominal aorta containing the bullet just above the bifurcation was excised. Complete recovery followed. The bullet apparently entered the heart or was carried there from one of the great vessels, and remained there for twenty-six days.

The following are ten fatal cases of thoracic bullet wounds with peripheral arterial embolization of the missile.

CASE 7.—Schmidt,[9] 1885. A man, aged 30 years, committed suicide. The body was studied in an anatomic laboratory, and no details of the clinical course are available. The bullet was 3 cm. long, 2 cm. in circumference and weighed 3.45 Gm. It entered the sixth interspace 10 centimeters to the left of the sternum, passed upward and through the anterior wall of the left pulmonary vein, and caused slight damage to the posterior wall. It was then carried by the blood stream into the left auricle through the left ventricle and became lodged in the left femoral artery, where it was associated with a thrombus 15 cm. long, indicating a period of survival. The cause of death was thought to be pulmonary hemorrhage, and the author states that he had not found a similar case previously reported.

CASE 8.—Dittrich,[10] 1897. The author reports only the following details which were apparently found at autopsy following suicide. There was a conical bullet low in the abdominal aorta. It had penetrated the anterior wall of the ascending aorta, reached the aortic lumen, and probably descended only on the manipulation of the cadaver during transportation.

CASE 9.—O'Neill,[11] 1917. A soldier sustained a wound from a shell fragment in the left posterior axillary line at the level of the fifth interspace. Roentgenograms three days later showed paralysis of the left half of the diaphragm, a left pleural effusion, and a foreign body probably in the left ventricle. A half hour after the roentgenograms the left leg became cold and cyanotic from the knee to the ankle. Roentgenograms on the fourth day showed the foreign body to be absent from its previous thoracic location, but present in the midabdomen apparently at the aortic bifurcation. The patient died on the fifth day of "generalized gas gangrene," and at autopsy the bullet was found at the bifurcation of the left common iliac artery. The pericardium had a small tear with a small amount of blood in it. A tear of the interventricular septum and of the cordae tendinae of the aortic valve indicated probable entrance into the left ventricle.

CASE 10.—De Rouvault,[12] 1917. A soldier sustained a bullet wound, caliber not stated, at the external end of the spine of the right scapula. There was shock, hemoptysis, a right hemothorax, and motor and sensory paralysis of the left leg. A roentgenogram showed no foreign body in the spinal, thoracic, or abdominal regions, but a roentgenogram on the fourth day showed the bullet in the region of the left femoral artery. The treatment was supportive, and the bullet was removed on the fourth day from the left femoral artery under local anesthesia. The patient died on the sixth day and autopsy showed a right hemothorax, congestion of the base of the left lung, a transverse wound of the lower lobe of the right lung, and gangrene of the left leg. The aortic wound was an irregular triangular defect at the junction of the arch and the descending aorta. Its greatest dimension was 5 millimeters on the outside and 15 millimeters on the inside. A small superficial wound of the intima was present on the opposite wall. There was a slight periaortic effusion of blood.

CASE 11.—Bland-Sutton,[13] 1919. A soldier was struck in the "chest" by a rifle bullet, caliber not stated. The bullet was palpated in the left external iliac artery before death. A thoracentesis was done. Four pints of "fused blood" were recovered. The bullet wound was found in the left external iliac artery at autopsy, and there was a "small wound in the aorta just below the arch." Death was thought to be due to streptococcal infection as a positive culture was obtained.

CASE 12.—LeClerq and Muller,[14] 1930. A man, after being wounded by an 8 millimeter bullet, 3 centimeters to the left of the midline of the back, at the level of the eighth dorsal vertebra, brought up "frothy blood" and died after walking "a few steps." Autopsy showed the bullet had passed through the muscles, grooved the left surface of the seventh dorsal vertebrae, passed through the thoracic aorta, pierced the left bronchus but could not be found anywhere in the thoracic viscera. There was hemorrhage into the posterior mediastinum down to the diaphragm. The bullet was found in the left femoral artery where the profunda arises. Apparently the bullet fell back into the aorta after causing an opening in the left bronchus.

CASE 13.—LeClerq and Muller,[14] 1930. A woman, aged 27 years, sustained a wound from a "ball," size not stated, fired from a distance of about 6 meters. It pierced the upper part of the left thorax near the axillary line. The survival period was not stated, but autopsy the "next day" disclosed the bullet had pierced the left lung causing abundant hemorrhage into the pleural cavity, pericardium, and mediastinum. There was erosion of

The very interesting experimental work of Fritz and co-workers[26] indicates exceedingly well what may be expected when foreign bodies gain entrance directly or indirectly to the cavities of the heart. Fritz and his associates introduced foreign bodies into the left side of the heart through the left auricular appendage in thirteen dogs. In twelve animals the foreign body was immediately embolized to the aorta or one of its branches. In one animal the foreign body was trapped in the auricle by the ligature used to close it. Of fifteen foreign bodies introduced into the right ventricle thirteen were promptly carried into the pulmonary circuit; one was caught behind the papillary muscles of the right ventricle. No cardiac dysfunction resulted. Autopsy in this animal ten days later showed the foreign body to be encapsulsated by a thin layer of fibrin with no associated thrombus formation. In the other animal, the foreign body remained in the tip of the right auricle and was found to be firmly fixed there without a propagating thrombus at autopsy twelve weeks later.

Foreign bodies were introduced into the jugular vein in seven dogs and in all embolized to the lungs. In a few of the animals the foreign body remained in the chambers of the heart for as long as thirty-six hours.

One would expect a foreign body entering the aorta or left heart chambers to be propelled quickly in the direction of blood flow. However, this did not occur in the case reported by Tegeler.[27] The patient recovered and had no cardic symptoms for thirteen months when he was accidentally electrocuted. Autopsy disclosed that the bullet had entered the aorta, fallen back into the left ventricle where it became encapsulated by fibrin, and was held in place by the chorda tendineae. In Singleton's[28] case a farm laborer was shot accidentally in the anterior part of the left chest. A "state of collapse" followed but the patient returned to work in four days. He died suddenly on the fifty-fourth day after the injury. At autopsy the bullet was found encapsulated in the left ventricle by a thin coat of fibrin, having passed through the right ventricle, the upper portion of the septum, the semilunar valve, and the aortic wall before entering the left ventricle.

SUMMARY

A case of thoracic bullet wound followed by embolization to the left femoral artery is presented. This appears to be the sixth surviving case of its kind, five others having been found in the literature. In addition fifteen other cases in which bullet wounds of the heart or associated great vessels had been followed by peripheral arterial embolization of the foreign body are summarized.

It is obvious from the behavior of the missile both in the clinical cases and in animal experiments that any conclusion as to the point of entrance into the vascular system on the basis of time of embolization is unsound. The entrance of a missile into the cardiovascular system through the wall of a heart chamber or aorta might be determined with greater accuracy on the basis of the presence or absence of hemoptysis, pericardial friction rub, or effusion, murmurs, electrocardiographic changes, and the results of fluoroscopic and x-ray studies.

Detailed study of these cases suggests that a thoracic wound with a small caliber missile may do no serious damage to the lungs and pulmonary vessels but still may have sufficient force to pierce one wall of the aorta or of a cardiac chamber. It may then be embolized immediately or later to the systemic vessels, being carried usually to the iliac or femoral vessels and more commonly to the left side. The path of a bullet which appears to afford the best chance of entering the aorta without damage to associated structures is apparently through the anterior chest wall to the left of the sternum. A wound involving the abdominal aorta is likewise compatible with survival for a time, but is usually associated with extensive damage to other structures leading to higher mortality, *there being no reported cases of survival from this combination of injuries.*

Therefore, in instances of bullet wounds with only a wound of entrance, failure to demonstrate the foreign body in the expected region should prompt the suspicion that it may have entered the cardiovascular system and been carried away, thus suggesting that other parts of the body be studied roentgenographically. Early motor and sensory paralysis due to ischemia may suggest the erroneous diagnosis of a lesion of the nervous system. Finally, the correct diagnosis should lead to early embolectomy, a recommendation made by Schloffer in 1903. With the present improvements in the technique of embolectomy, recoveries from aortic or cardiac wounds with embolization of the foreign body might, in the future, be complete even to the survival of the involved extremity.

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THE EFFECT OF DECREASING THE AMOUNT OF LUNG TISSUE ON THE RIGHT VENTRICULAR PRESSURES IN ANIMALS*

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STUDIES of the dynamics of the lesser circulation have been carried out by many workers since at least 1850. Most of the studies reported have been acute experiments in which the pulmonary vascular bed was gradually occluded. Right auricular or ventricular, or pulmonary artery pressures along with systemic arterial and venous pressures were recorded. The pressures in the lesser circulation were recorded by means of placing a glass tube directly into a branch of the pulmonary artery (Beutner, 1852),[1] or by passing a glass or silver tube through an external jugular vein into the right heart as was reported by Badoud[2] in 1874.

Lichtheim,[3] doing experiments on dogs, using curare and artificial respiration, in Cohnheim's and Heidenhain's laboratories, reported in 1876 that up to three-fourths of the pulmonary vascular bed could be occluded without changes occurring in the carotid or peripheral venous pressures. The mean pulmonary arterial pressure rose as he obstructed more and more of the pulmonary vascular bed. When more than three-fourths of it was occluded, the systemic pressure began to fall, although the mean pulmonary arterial pressure remained constant or rose slightly. If still more of the vascular bed were obstructed, the animal soon died. Welch,[4] Plumier,[5] Tigerstedt,[6, 8] and Gerhardt[7] confirmed these results. Scarff[9] measured the pulmonary arterial pressure in dogs during pneumonectomy. Gibbon and co-workers[10] found that there was no significant effect on arterial and venous pressures until more than 60 per cent of the pulmonary artery was occluded. Fineberg and Wiggers[11] studied the diastolic-systolic right ventricular and aortic pressures in dogs. They concluded that compensatory changes occur in the right heart sufficient to prevent changes in the aortic pressure pulses, up to a point where roughly 58 per cent of the pulmonary circulation was occluded.

Long and co-workers[12] found that if in the dog one lung root is suddenly occluded, a rise occurs in the right ventricular pressure which reaches a peak in two to ten minutes and then falls slowly to return to the pre-ligation level in twenty-five to thirty minutes in most cases.

Since the advent of cardiac catheterization in man, the dynamics of the lesser circulation have been studied in patients with a wide variety of various

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**U. S. Public Health Service Research Fellow of the National Institutes pulmonary and cardiac disorders. Cournand[13] and co-workers studied pulmonary hemodynamics before and after pneumonectomy. Mendelsohn[14] and co-workers studied the pulmonary arterial pressure during surgery in a group of eleven patients subjected to various chest operations, including pneumonectomy.

Although it is well known that most patients are able to tolerate a pneumonectomy with surprisingly few obvious circulatory changes, a certain number of patients—notably those in the older age group—are not able to withstand the rigors of a pneumonectomy. In some cases this may be due to the failure of the right ventricle to make a satisfactory adjustment to the strain of having the pulmonary vascular bed suddenly reduced by 50 per cent.

It was thought, therefore, that it would be of interest to study the right ventricular pressures in a controlled animal experiment during and after the removal, in stages, of up to 85 per cent of the pulmonary tissue by a combination of pneumonectomy and lobectomy. The question arose as to whether there might be some correlation between the pressure changes and the survival or death of the animal when the pulmonary tissue was reduced to very low levels.

It is the purpose of this paper to report our observations on the right ventricular pressures during operation; (1) in a group of animals undergoing pneumonectomy; (2) in a group undergoing lobectomy or bilobectomy following pneumonectomy; and (3) in a group where the lung tissue was reduced to one or two lobes by three operations. The right ventricular pressures were determined at intervals following operation in most of the surviving dogs. Also systemic pressures, blood oxygen analysis, and hematocrits were studied.

TECHNIQUE

Normal, healthy mongrel dogs weighing from 10 to 14 kilograms were used. They received 45 mg. of morphine as premedication. Under local anesthesia, an 8F cardiac catheter was introduced, in most cases through one of the external jugular veins into the right ventricle, under fluoroscopic control. Occasionally a femoral vein was used. The right ventricle, rather than the pulmonary artery, was chosen for the measurements for several reasons. It was found that it was easier to maintain the position of the catheter in the ventricle rather than in the artery because of the manipulations taking place before and during the operation. Moreover, it is not always easy to place the tip of the catheter in a given portion of the artery; for instance, the catheter might be located in the artery to be divided. Also artefacts are more common in recording pulmonary arterial pressures than ventricular pressures. Very often some disturbance in rhythm occurred as the catheter was being manipulated into the ventricle, but once it was in place, no further disturbance was observed. Difficulty because of having the tip of the catheter in the right ventricle during the operation was not experienced. Because of convenience, heparin 0.5 mg. per c.c. rather than a saline drip was used to keep the catheter open. The total amount of heparin rarely exceeded 5 mg., and this caused no unusual bleeding during the surgery. Measurements were made either with a Hamilton manometer or a Sanborn Electromanometer, using an electrocardiogram apparatus for recording.

Fig. 1 shows an anteroposterior and a lateral roentgenogram of a normal dog with the cardiac catheter in place.

Recordings of heart rate and right ventricular pressures were made in the following order: while the animal was still awake; after induction of ether anesthesia; after insertion of an endotracheal tube and connection with the anesthesia machine; after the chest was opened; after the artery to the lobe or lung to be removed was mobilized; *immediately* after ligation of the artery; after lobectomy or pneumonectomy; after closure of the chest; and at the conclusion of the procedure with the anesthetic discontinued, as may be seen in the tables.

The anesthesia used for the first operation was ether and air given under slight continuous positive pressure. Ether-oxygen was used for the second and third stages, with controlled respiration while the chest was open. With this method, the animals practically always remained in good condition during the operation, even when the left upper lobe was the only remaining lung tissue.

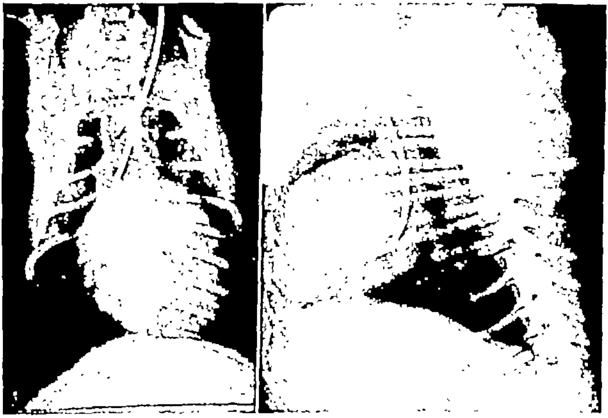


Fig. 1.—Anteroposterior and lateral chest roentgenogram of normal dog showing cardiac catheter in place.

The operations were all done by the usual dissection technique. After completion of the procedure an attempt was made to adjust the intrapleural pressure to about -15 to -5 cm. of water. The opening in the jugular vein was then closed with 00000 deknatel suture to save it for subsequent catheterizations. After the first operation the animal received no special care. After the second or third, the animal was placed in an oxygen box for a few hours. Penicillin was used only exceptionally.

Following the first stage, at least three months were allowed to elapse before further reduction of the pulmonary tissue was carried out in order that the animal might recover fully. In one group of animals, measurements were

TABLE I. RIGHT VENTRICULAR PRESSURES IN MM. OF HG
LEFT OR RIGHT PNEUMONECTOMY

DOG NO.	DOG AWAKE	DOG ASLEEP	POSITIVE PRESSURE	CHEST OPEN	ARTERY ISOLATED	ARTERY TIED	AFTER PNEUMONECTOMY	CHEST CLOSED	ANESTHESIA STOPPED	LIVING OR DEAD
4	40/3	43/0	42/2	41/3	---	45/7	50/7	47/3	43/3	L
36	30/3	34/2	29/0	30/0	33/-2	50/2	50/1	44/12	40/11	L
242	40/1	38/-2	35/2	34/4	43/7	54/10	53/8	45/5	45/2	D
379	33/4	30/4	32/3	35/10	37/3	54/5	56/3	62/2	64/1	L
435	26/2	32/0	36/5	34/5	32/4	42/5	57/3	51/3	51/3	L
436	35/7	33/18	37/22	35/8	37/10	47/11	52/9	57/11	54/8	L
437	34/10	37/6	32/8	30/7	33/8	39/9	58/6	36/5	43/4	L
468	---	---	50/10	42/7	---	--	---	---	42/0	L
494	---	20/1	31/-2	44/3	---	40/10	48/7	57/7	30/3	D
539	40/5	35/5	37/5	38/2	48/4	68/8	54/10	52/12	48/-2	L
865	26/10	30/11	35/2	32/4	35/6	40/6	40/5	40/6	39/6	L
923	25/5	---	28/3	25/0	25/-2	26/-6	30/0	25/10	---	L
924	---	32/6	30/3	32/7	30/4	39/5	48/8	39/2	33/4	L
925	25/5	26/6	---	28/5	32/3	40/10	42/11	---	---	L
985	25/1	22/-4	22/4	26/8	25/8	31/11	43/8	42/8	38/2	D
Av.	32/5	32/4	37/5	34/5	34/4	45/7	49/6	46/7	44/4	

Table II. Right Ventricular Pressures in Mm. Hg; Right Lower and Accessory Lobectomy in Dogs Having Previous Left Pneumonectomy

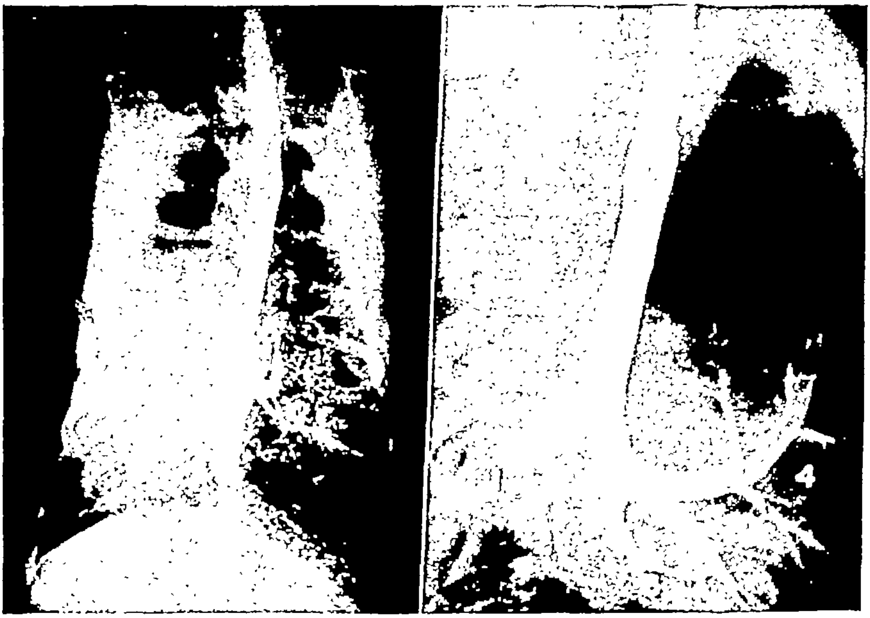
DOG NO.	DOG AWAKE	DOG ASLEEP	ON ANESTHETIC MACHINE	CHEST OPEN	ARTERY ISOLATED	ARTERY TIED	AFTER LOBECTOMY	CHEST CLOSED	ANESTHESIA STOPPED	WEEKS	POST-OPERATIVE	LIVING OR DEAD
9	42/4	45/2	44/2	42/5	44/8	64/12	68/9	78/2	78/6	--	---	D
69	33/8	38/2	42/0	42/0	43/1	75/5	85/8	75/0	75/2	--	---	D
468	---	29/8	30/10	55/10	---	76/10	68/9	47/8	41/5	--	---	D
478	42/3	37/2	25/2	34/4	33/5	38/8	48/4	42/4	42/6	--	---	D
530	58/6	48/3	46/4	48/4	48/5	62/6	80/5	72/2	71/3	--	---	D
617	23/2	29/4	26/6	27/8	28/7	48/8	58/8	50/2	52/2	--	---	D
673	---	30/3	31/5	26/6	36/8	57/12	50/17	44/21	36/12	--	---	L
810	30/2	42/16	36/2	38/5	31/0	50/5	58/5	52/0	51/0	6	36/16	L
855	44/5	46/−2	33/0	30/−2	32/4	38/3	38/2	38/0	35/0	34	60/3	L
857	38/11	35/8	37/3	36/5	38/5	56/6	64/5	58/5	62/4	5	41/5	L
										14	44/8	
901	43/7	34/6	43/4	19/5	31/10	46/17	43/14	62/7	64/6	37	48/6	L
911	34/1	35/1	34/0	30/2	28/3	32/6	58/6	60/3	60/4	--	---	D
924	44/6	38/5	41/5	37/6	30/10	77/10	85/7	90/13	92/12	2	74/3	L
										30	81/7	
925	32/4	38/6	34/3	32/10	37/8	53/11	52/8	68/2	72/1	2	45/3	L
										6	42/−2	
										30	55/4	
936	37/4	28/2	35/0	32/1	36/0	61/2	61/4	44/1	50/0	38	43/10	L
Av.	40/5	37/5	36/3	35/5	36/6	56/8	61/7	59/5	59/4			

taken during each stage; in another group, only during the second or second and third operations.

Fig. 2 shows a bronchogram of the remaining lung tissue in a dog that had undergone pneumonectomy and right lower and accessory lobectomy.

RESULTS

Table I shows the measurements which were taken before, during, and after pneumonectomy in a group of fifteen animals. All animals survived the procedure, three dying later of miscellaneous causes. Nine of these have undergone further reduction in lung tissue. It can be seen that most of the dogs followed the average of the group rather closely. Up to the point just prior to ligation of the artery there was very little change from the initial pressures. For some unexplained reason, Dog 436 had temporarily a very high diastolic pressure. At the point where the artery was ligated, it can be seen that there was a slight to moderate rise in the systolic component, averaging 10 mm. Hg.



A. B.

Fig. 2.—*A*, Bronchogram showing entire chest of animal having only the right upper and middle lobes remaining. *B*, Spot film of same animal. 1. Stump of left main bronchus. 2. Stump of bronchus transected above lower and accessory lobe bronchi. 3. Right middle lobe. 4. Right upper lobe.

The systolic pressure was still a little higher in most of the dogs by the time removal of the lung was completed. At the end of the procedure the systolic pressure was somewhat lower in most of the dogs, although it still averaged 9 mm. higher than initially. There was little change in the diastolic pressure in this group during the entire procedure.

Table II shows the measurements of fifteen animals which were taken at the time of removal of the right lower and accessory lobes. All these animals had had a previous left pneumonectomy. All animals survived the operation, four died within twenty-four hours, and three others between one week and several months later of causes other than cardiorespiratory failure. Eight are still living for periods up to eighteen months. The initial systolic pressure was moderately elevated in several of the animals. As with the pneumonectomy group, however, there was, with a few exceptions, no great change in pressure until ligation of the artery. When the artery was ligated, an *immediate* average rise of 20 mm. Hg occurred in the systolic pressure, with a variation of from 4 mm. to 38 mm. Hg. After the removal of the lung, the average systolic pressure was 5 mm. higher. Little change in pressure was observed from then on until after the anesthetic was stopped. Several of the dogs exhibited a high diastolic pressure at some period during the surgery, but the diastolic pressure for the group remained almost constant throughout.

Table III shows the measurements which were taken at the time of removal of the left lower lobe in eight animals. These dogs had all had the right lung removed previously. Four of these animals died within twenty-four hours of pulmonary edema, two died a few days later of causes not directly related to the small amount of lung tissue remaining, and two are still surviving. These dogs consistently had, prior to ligation of the artery, a higher systolic pressure than the group of animals in Table II. After ligation of the artery the systolic pressure rose an average of 14 mm. Hg, with a variation between 6 and 28 mm. Hg. The systolic pressure rose an average of 8 mm. Hg more by the time the lobe was removed, and had fallen 3 mm. by the time the anesthesia was discontinued. It may be noted that the diastolic pressure was high in this group after ligation of the artery, and remained elevated at the conclusion of the procedure.

Table IV shows the measurements of five animals, four in which the lung tissue was reduced to the left upper lobe, and one to the right upper and middle lobes, by three operations. It was thought that there might be a lower mortality rate with a more gradual reduction in pulmonary tissue. The first four animals all died, two within twenty-four hours, and two later. As the results from these few animals did not seem to indicate any advantage in the three-stage procedure, it was abandoned.

Measurements taken at intervals following surgery in the survivors show right ventricular systolic pressure to be lower than at the end of the operation in most of the animals, but higher than in normal dogs. In several it is very high.

The results of systemic pressures taken before and after surgery showed that the pressure in most cases was not greatly different after the surgery than initially. In general, it tended to be a little higher; significantly lower values were found in none.

The hematocrits taken before, during, and after the procedures did not show any significant change. Except for the fact that the heart rate always rose after induction of anesthesia, no constant changes were noted.

In the surviving animals blood oxygen analyses have shown a normal value in almost every case.

Table III. Right Ventricular Pressures in Mm. Hg; Left Lower Lobectomy in Dogs Having Previous Right Pneumonectomy

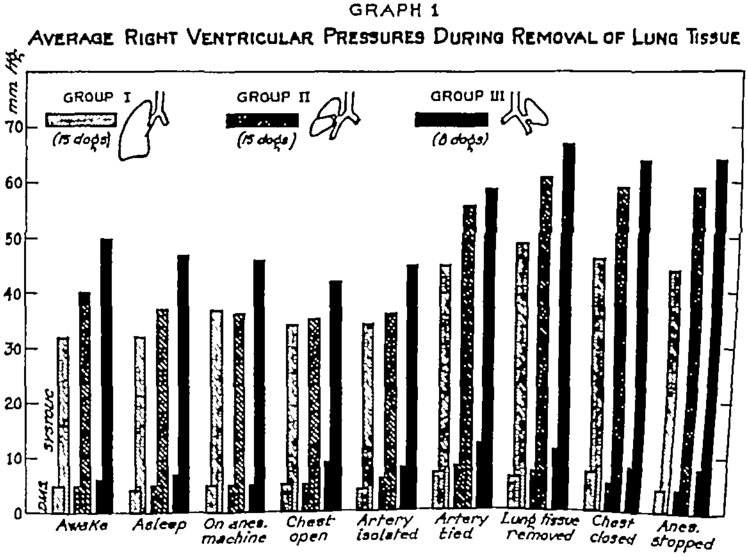
DOG NO.	DOG AWAKE	DOG ASLEEP	ON ANESTHETIC MACHINE	CHEST OPEN	ARTERY ISOLATED	ARTERY TIED	AFTER LOBECTOMY	CHEST CLOSED	ANESTHESIA STOPPED	WEEKS	POST-OPERATIVE	LIVING OR DEAD
4	---	40/1	43/6	44/5	45/8	52/12	58/18	54/8	55/8	--	---	D
36	43/2	45/–2	50/–2	42/4	51/3	80/9	56/9	80/2	80/0	5	43/–2	L
										30	70/6	
379	48/4	41/5	44/6	46/12	48/12	56/15	52/6	55/6	60/9	--	--	D
394	48/8	---	48/4	42/5	48/8	76/15	86/18	73/10	74/12	--	--	D
436	48/4	34/17	40/6	34/12	41/12	54/16	90/11	70/16	63/8	--	--	D
437	53/11	58/10	52/5	38/10	36/11	42/14	72/11	63/8	64/8	--	--	D
486	63/8	59/7	53/6	43/15	46/8	60/9	63/8	68/6	63/8	--	--	L
593	---	42/2	41/5	45/6	42/5	53/8	55/8	52/9	53/8	--	--	D
Av.	50/6	46/7	46/5	42/9	45/8	59/12	67/11	64/8	64/8			

Table IV. Right Ventricular Pressures in Mm. Hg; Reduction of Lung Tissue in Three Stages

DOG NO.	STAGE NO.	DOG AWAKE	DOG ASLEEP	ON ANESTHETIC MACHINE	CHEST OPEN	ARTERY ISOLATED	ARTERY TIED	AFTER OPERATION	CHEST CLOSED	ANESTHESIA STOPPED	WEEKS	POST-OPERATIVE	LIVING OR DEAD
256	II	---	48/22	40/4	40/10	56/10	76/10	48/20	48/5	48/4	---	---	
	III	44/10	52/7	38/9	35/12	54/10	53/10	67/18	---	---	---	---	D
680	II	---	30/1	28/4	30/3	33/3	36/5	38/8	34/18	30/14	---	---	
	III	56/8	42/10	42/7	40/14	40/16	46/20	46/24	47/20	48/24	40	60/4	L
26	II	35/11	35/10	30/12	30/14	36/17	40/20	44/2	48/20	44/18	---	---	
	III	60/4	---	64/13	74/9	66/20	90/18	120/42	74/10	71/10	---	---	D
104	I	46/3	46/2	40/0	44/3	47/5	53/4	54/10	58/8	47/5	---	---	
	II	32/0	48/7	28/2	23/0	20/0	36/0	34/1	40/0	38/0	---	---	
	III	40/0	38/–2	36/0	34/12	50/7	60/8	58/8	78/9	74/8	---	---	D
999	I	26/3	27/12	32/10	29/13	34/17	38/1	42/5	40/5	38/2	---	---	
	II	55/6	---	72/10	55/11	56/10	78/12	---	---	---	---	---	
	III	48/2	36/0	46/1	56/8	62/5	65/3	72/4	68/10	74/11	---	---	D

DISCUSSION

This approach to the problem of the changes occurring in the lesser circulation when a portion of the pulmonary vascular bed is occluded is different from most of the previous work; lung tissue was removed, and the animals kept alive. Previous work has largely consisted of acute experiments with partial or complete obstruction of the pulmonary artery being carried out over periods varying from a few minutes to several hours. We realize, of course, that reducing the lung tissue in stages gives the heart and the remaining pulmonary vascular bed time for adjustment. Sudden occlusion of all the arteries except to one lobe is not the same as gradual reduction as we have carried out. Also, as the mediastinum is not a stable structure in dogs, the remaining lung tissue will expand until it fills the space previously occupied by both lungs.

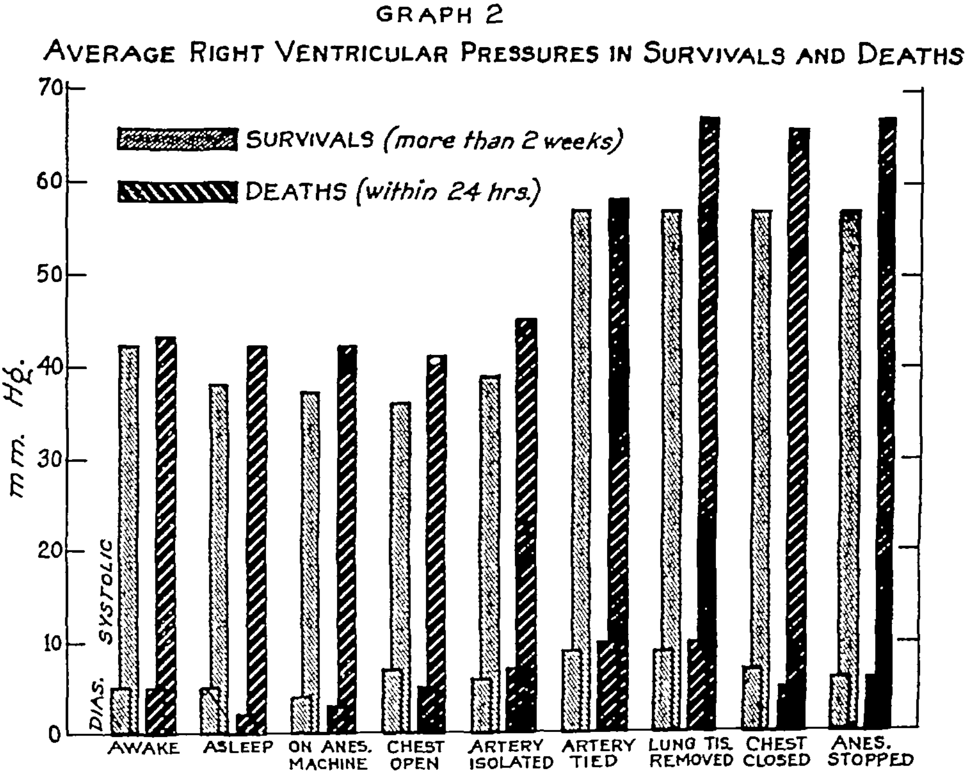


Graph 1.

Whether or not the animal survives the reduction of this amount of lung tissue depends not only on the ability of the right heart to adjust to the increased resistance, but also on whether enough pulmonary tissue remains to supply the respiratory needs of the animal. Of the seventeen deaths occurring in the group of dogs that had a second or third operation, ten occurred within a twenty-four hour period, seven occurred from a few days to several months later of miscellaneous causes. The ten dogs that died soon after operation all exhibited a similar picture of increasingly severe dyspnea and cyanosis. At autopsy all similar prominent finding—massive pulmonary edema. Gross dilatation of the heart was observed in several of these animals, but not all.

It is difficult to separate the cardiac from the pulmonary factors in these animals. However, as soon as pulmonary edema appears, a vicious circle is set up. The remaining lung tissue is less able to oxygenate the blood, causing increasing damage to the pulmonary tissue and myocardium with more pulmonary edema until the animal dies.

The surviving animals all appear surprisingly healthy, although several of them have a very high right ventricular systolic pressure, indicating that the increased resistance caused by the decreased vascular bed has imposed considerable strain on the right ventricle. It would appear that the right ventricle has a considerable ability to compensate; that is, it is able to overcome the increased resistance without any obvious ill effects, at least for many months.


GRAPH 2

AVERAGE RIGHT VENTRICULAR PRESSURES IN SURVIVALS AND DEATHS

Graph 2.

One sees from Graph 1 that a definite difference existed in the systolic pressures in the three groups of animals, which goes along with the amount of reduction in the pulmonary vascular bed. It is not clear just why there was the observed difference between groups 2 and 3 up to the point of ligation of the artery. Up to this point the only difference in the amount of reduction was that the dogs in group 2 had a left pneumonectomy, while those in group 3 had a right pneumonectomy. Although the right lung is somewhat larger than the left, it is questionable whether this is the only factor accounting for the observed difference. After ligation the amount of increase in pressure would appear to correlate rather well with the amount of reduction of the vascular bed. The diastolic pressure in group 3 was well above that in group 2, especially after ligation of the artery, being considerably above normal in many of the dogs in group 3. This may indicate that the right heart was very nearly on the verge of decompensation, when this amount of reduction was carried out.

From this graph, it is apparent that at several points the animals in group 1 had slightly higher pressures than those in group 2. The fact that group 1 had continuous positive pressure anesthesia might account for this.

From Graph 2, which shows the average pressures of the dogs dying within twenty-four hours and those surviving the last operation by at least two weeks, one gets the impression that the dogs which died had somewhat higher pressures than those that survived. However, the individual variations are such that this is probably of little significance.

It is apparent from the tables that the mortality in the group having only the left upper lobe remaining was much higher than in the group where the right upper and middle lobes were the only remaining lung tissue. It is probable that the difference in the amount of remaining lung tissue in the two groups accounted for this. It is also probable that in most dogs the point of maximum reduction compatible with life lies just between these two groups.

The follow-up pressures in the surviving animals indicate, as one might expect, that in most cases the systolic pressure falls as the remaining pulmonary vascular bed becomes adjusted to the increased blood flow through it. However, most of the pressures remain at levels higher than normal, indicating an increased resistance to the right heart output.

SUMMARY AND CONCLUSIONS

1. Data are presented on changes occurring in the right ventricular pressure in dogs during removal of lung tissue.

2. In the majority of animals decreasing the pulmonary tissue caused a rise in the right ventricular systolic pressure somewhat in proportion to the amount removed.

3. Many dogs tolerated reduction to the right middle and upper lobes. Very few were able to tolerate removal of all pulmonary tissue except the left upper lobe. If the animal survived the first twenty-four hours following operation, the chance of prolonged survival was good.

4. It was difficult to correlate the pressure changes with the survival or death of the animal.

5. The right ventricle in dogs has considerable power of compensation, providing the stress is imposed gradually.

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INDEX TO VOLUME 21

A

Ackerman, Lauren (with Bosher, Lewis H., Jr., and Burford, Thomas H.) Pathology of experimentally produced lye burns and strictures of esophagus, 483
  (with Jackson, Elisabeth, and Bertoli, Ferruccio) Exfoliative cytology—an adjunct in diagnosis of bronchiogenic carcinoma, 7
  (with O'Neal, Lawrence W.) Cartilaginous tumors of ribs and sternum, 71
Ada, Alexander, E. W. (with Hevenor, Engel P.) Reconstruction of defects of thoracic wall with tantalum mesh gauze, 125
Adams, W. E. (with Carlson, Ralph F., Charbon, Bernard C., and Charbon, Harmin G. A.) Effect of decreasing amount of lung tissue on right ventricular pressures in animals, 621
Adenoma, bronchial—analysis of 26 cases (Delarue), 335
    (Doty, Robert D.), 340
  in supernumerary tracheal lobe, bronchial (Epstein), 362
Adhesions in opaque lobes, observations on division of (Coello), 135
American Association for Thoracic Surgery
  List of members, 634
  List of officers, council, membership committee, 633
  Thirty-first annual meeting—program, 420
Anastomosis of blood vessels, clamps for end-to-end (Charbon), 109
Anesthetic explosions (Buckles), 426
Aneurysm with sternal perforation, successful repair of an aortic (Cowley, Sloan, and Sullenberger), 159
Angiocardiographic findings in thoracoplasty, artificial pneumoperitoneum, and phreniclasia (McCoy, Steinberg, and Dotter), 149
Angiocardiography, diagnostic value of dynamic studies in—evaluation of new rapid technique, (Keyes, Wegelius, and Lind), 164
Anomalous lobe of lung arising from esophagus (Gans and Potts), 313
  origin of right subclavian artery from descending aorta, report of two cases of (Sealy), 319
Anomaly of pulmonary veins (Cooke, Evans, Kistin, and Blades), 452
Aorta at level of diaphragm treated successfully with preserved human blood vessel graft, coarctation of (Beattie, Cooke, Paul, and Orbison), 506
Aorta—Cont'd
  in order to reach bifurcation of trachea, mobilization of arch of (Monod, Babou, Kateb, and Ghazi), 492
  report of two cases of anomalous origin of right subclavian artery from descending (Sealy), 319
Artery from descending aorta, report of two cases of anomalous origin of right (Sealy), 319
  internal costal branch of internal mammary (Kropp), 421
Ashe, William M. (with McDonald, John R., and Clagett, O. Theron) Nonspecific pneumonitis of left upper lobe (simulating the "middle lobe syndrome" and producing early superior pulmonary sulcus syndrome), 1
Aspiration through endotracheal tube and extubation, sudden death occurring immediately after operation in patients with cardiac disease, with particular reference to role of (Shumacker and Hampton), 48
Atresia of esophagus treated by one-stage primary esophagogastrostomy employing right transpleural approach, 460

B

Babou, G. (with Monod, Olivier, Kateb, E., and Ghazi, S.) Mobilization of arch of aorta in order to reach bifurcation of trachea, 492
Beattie, Edward J., Jr. (with Cooke, Francis N., Paul, John S., and Orbison, James A.) Coarctation of aorta at level of diaphragm treated successfully with preserved human blood vessel graft, 506
Beatty, Arch J. (with Buckingham, William W., and Ottosen, Poul), Resection Versus thoracoplasty in treatment of pulmonary tuberculosis, 34
  (with Ottosen, Poul, Popp, Carol, and Buckingham, William W.) One hundred thirty consecutive thoracoplasties—evaluation of results in different types of pulmonary tuberculosis, 202
Bertoli, Ferruccio (with Jackson, Elisabeth, and Ackerman, Lauren V.) Exfoliative cytology—an adjunct in diagnosis of bronchiogenic carcinoma, 7
Blades, Brian (with Cooke, Francis N., Evans, John M., and Kistin, Albert D.) Anomaly of pulmonary veins, 452

Blakemore, William S. (with Fowler, Ward S.) Lung function studies—VII. Effect of pneumonectomy on respiratory dead space, 433
Blalock, Alfred. Consideration of some of problems in cardiovascular surgery, 543
Blood vessels, clamps for end-to-end anastomosis of (Charbon), 109
  volume and extracellular fluid in tuberculosis (Hay and Jenkins), 42
Bone, monostotic fibrous dysplasia of (Flickinger), 298
Bosher, Lewis H., Jr. Review of surgically treated lung abscess, 370
  (with Burford, Thomas H., and Ackerman, Lauren) Pathology of experimentally produced lye burns and strictures of esophagus, 483
  (with Taylor, Frederick H.) Heterotopic gastric mucosa in esophagus with ulceration and stricture formation, 306
Boyden, E. A. (with Hamre, C. J.) An analysis of variations in bronchovascular patterns of middle lobe in fifty dissected and twenty injected lungs, 172
Bradshaw, H. H. (with Myers, Richard T.) Benign intramural tumors and cysts of esophagus, 470
Brantigan, O. C. Oxygen administration: an emergency method when intratracheal tube fails with chest open, 532
Bronchial adenoma—analysis of 26 cases (Delarue), 535
    in supernumerary tracheal lobe (Epstein), 362
    nomenclature and present methods of treatment (Doty), 349
  closure, effective method for (Rasmussen), 513
  defects with stainless steel wire mesh, experimental reconstruction of tracheal and (Bucher and Burnett), 572
  yardstick (Emerson), 516
Bronchiectasis, pulmonary artery in (Gobbel, Gordon, and Digman), 385
Bronchiogenic carcinoma, an adjunct in diagnosis of—exfoliative cytology (Jackson, Bertoli, and Ackerman), 7
  neoplasm masquerading as alveolar cell carcinoma (Potts and Davidson), 402
Bronchoscopy in treatment of spontaneous and traumatic pneumothorax (Rubin and Rubin), 377
Bronchovascular patterns of middle lobe in fifty dissected and twenty injected lungs, an analysis of variations in (Boyden and Hamre), 172
Bucher, Robert M. (with Burnett, W. Emory) Experimental reconstruction of tracheal and bronchial defects with stainless steel wire mesh, 572
Buckingham, William W. (with Beatty, Arch J., and Ottosen, Poul) Resection versus thoracoplasty in treatment of pulmonary tuberculosis, 34
Buckingham, William W.—Cont'd
  (with Ottosen, Poul, Popp, Carol, and Beatty, Arch J.) One hundred thirty consecutive thoracoplasties—evaluation of results in different types of pulmonary tuberculosis, 202
Buckles, Maurice G. Anesthetic explosions, 426
Bullet embolus to left femoral artery following thoracic gunshot wound (Keeley), 608
Burford, Thomas H. (with Bosher, Lewis H., Jr., and Ackerman, Lauren) Pathology of experimentally produced lye burns and strictures of esophagus, 483
Burnett, W. Emory (with Bucher, Robert M.) Experimental reconstruction of tracheal and bronchial defects with stainless steel wire mesh, 572

C

Carcinoma, bronchiogenic neoplasm masquerading as alveolar cell (Potts and Davidson), 402
  of esophagus, paraganglioma, simulating (Taylor), 189
Cardiac disease, with particular reference to role of aspiration through endotracheal tube and extubation, sudden death occurring immediately after operation in patients with (Shumacker and Hampton), 48
Cardiovascular surgery, consideration of some of problems in (Blalock), 543
Carlson, Ralph F. (with Charbon, Bernard C., Charbon, Harmia G. A., and Adams, W. E.) Effect of decreasing amount of lung tissue on right ventricular pressures in animals, 621
Carter, B. Noland (with Giuseffi, Jerome) Tracheotomy, useful procedure in thoracic surgery, with particular reference to employment in crushing injuries of thorax, 495
Cartilaginous tumors of ribs and sternum (O'Neal and Ackerman), 71
Charbon, Bernard C. Clamps for end-to-end anastomosis of blood vessels, 109
  (with Carlson, Ralph F., Charbon, Harmia G. A., and Adams, W. E.) Effect of decreasing amount of lung tissue on right ventricular pressures in animals, 621
Charbon, Harmia G. A. (with Carlson, Ralph F., Charbon, Bernard C., and Adams, W. E.) Effect of decreasing amount of lung tissue on right ventricular pressures in animals, 621
Child, Charles G., III (with Harmon, Glen S., Dotter, Charles T., and Steinberg, Israel) Liver herniation simulating intrathoracic tumor, 391
Citret, Coleman (with Juvenelle, Andrea A., Wiles, Charles E., Jr., and Stewart, John D.) Pneumonectomy with replantation of lung in dog for physiologic study, 111

Clagett, O. Theron (with Ashe, William M., and McDonald, John R.) Nonspecific pneumonitis of left upper lobe (simulating ''middle lobe syndrome'' and producing early superior pulmonary sulcus syndrome), 1
  (with Takaro, Timothy) Cavernous hemangioma of thoracic wall associated with clinical evidence of arteriovenous fistulas, 444
Clamps for end-to-end anastomosis of blood vessels (Charbon), 109
Clay, Richard C. (with Hanlon, C. Rollins) Pneumoperitoneum in differential diagnosis of diaphragmatic hernia, 57
Coarctation of aorta at level of diaphragm treated successfully with preserved human blood vessel graft (Beattie, Cooke, Paul, and Orbison), 506
Coello, Alfred J. Observations on division of adhesions in opaque lobes, 135
Collapse therapy of pulmonary tuberculosis, importance of extrapleural pneumothorax in (Cutler), 217
Congenital absence of pericardium (Shafiroff), 30
Cooke, Francis N. (with Beattie, Edward J., Jr., Paul, John S., and Orbison, James A.) Coarctation of aorta at level of diaphragm treated successfully with preserved human blood vessel graft, 506
  (with Evans, John M., Kistin, Albert D., and Blades, Brian) Anomaly of pulmonary veins, 452
Cowley, R. Adams (with Sloan, Herbert) Posterior mediastinal tracheotomy, 602
  (with Sloan, Herbert E., and Sullenberger, Neil H.) Successful repair of aortic aneurysm with sternal perforation, 159
Croxatto, Oscar C. (with Sampietro, Ruben) Pathology of pleural sclerosis: A study related to loss of expansivity of lungs and its treatment, 259
Cutler, J. W. Importance of extrapleural pneumothorax in collapse therapy of pulmonary tuberculosis, 217
Cysts of esophagus, benign intramural tumors and (Myers and Bradshaw), 470

D

Daniel, Rollin A., Jr. (with Diveley, Walter) Primary solitary neurogenic tumors of lung, 194
  (with Sappington, Thomas B., Jr.) Accessory diaphragm, 212
Daume, Robert (with Eaton, Robert M., Sherwood, J. Vincent, Kendrick, Pearl L., and Eldering, Grace) Serum protein levels and calculated osmotic pressures in tuberculosis: I. Correlation of values with severity of disease, 342
Davidson, Horace B. (with Potts, W. L.) Bronchiogenic neoplasm masquerading as alveolar cell carcinoma, 402
Delarue, N. C. Bronchial adenoma—analysis of 26 cases, 535
Diaphragm, accessory (Sappington, Thomas B., Jr., and Daniel, Rollin A., Jr.), 212
  treated successfully with preserved human blood vessel graft, coarctation of aorta at level of (Beattie, Cooke, Paul, and Orbison), 506
Diaphragmatic hernia, pneumoperitoneum in differential diagnosis of (Clay and Hanlon), 57
    with displaced liver, unusual (Knoepp), 304
Digman, George J. (with Gobbel, Walter G., Jr., and Gordon, Joseph) Pulmonary artery in bronchiectasis, 385
Diveley, Walter (with Daniel, Rollin A., Jr.) Primary solitary neurogenic tumors of lung, 194
Doppelt, Harry B. (with Goldberg, Jacob) Shoulder girdle dysfunction following thoracoplasty combined with partial scapulectomy, 584
Dotter, Charles T. (with Child, Charles G., III, Harmon, Glen S., and Steinberg, Israel) Liver herniation simulating intrathoracic tumor, 391
  (with McCoy, Herbert I., and Steinberg, Israel) Angiocardiographic findings in thoracoplasty, artificial pneumoperitoneum, and phreniclasia, 149
Doty, Robert D. Bronchial adenoma—nomenclature and present methods of treatment, 340
Dynamic studies in angiocardiography, diagnostic value of—evaluation of new rapid technique (Keyes, Wegelius, and Lind), 164

E

Eaton, Robert M. (with Sherwood, J. Vincent, Kendrick, Pearl L., Eldering, Grace, and Daume, Robert) Serum protein levels and calculated osmotic pressures in tuberculosis: I. Correlation of values with severity of disease, 342
Edwards, Jesse E. (with Peabody, Homer D., Jr., and Edwards, Jesse E.) Clinically indeterminate pulmonary fibrosis, a pathologic study, 519
Eldering, Grace (with Eaton, Robert M., Sherwood, J. Vincent, Kendrick, Pearl L., and Daume, Robert) Serum protein levels and calculated osmotic pressures in tuberculosis: I. Correlation of values with severity of disease, 342
Emerson, Ernest B., Jr. Bronchial yardstick, 516
Emphysema with defect of anterior mediastinum, obstructive (Lewis and Potts), 438

McDonald, John R. (with Ashe, William M., and Clagett, O. Theron) Nonspecific pneumonitis of left upper lobe (simulating "middle lobe syndrome" and producing early superior pulmonary sulcus syndrome), 1
Mediastinal tracheotomy, posterior (Adams and Sloan), 602
Mediastinum, obstructive emphysema with defect of anterior (Lewis and Potts), 438
Middle lobe in fifty dissected and twenty injected lungs, analysis of variations in bronchovascular patterns of (Boyden and Hamre), 172
"Middle lobe syndrome" and producing early superior pulmonary sulcus syndrome, simulating nonspecific pneumonitis of left upper lobe (Ashe, McDonald, and Clagett), 1
Moersch, Herman J. (with Peabody, Homer D., Jr., and Edwards, Jesse E.) Clinically indeterminate pulmonary fibrosis, pathologic study, 519
Monod, Olivier (with Babou, G., Kateb, E., and Ghazi, S.) Mobilization of arch of aorta in order to reach bifurcation of trachea, 492
Monostotic fibrous dysplasia of bone (Flickinger), 298
Myers, Richard T. (with Bradshaw, H. H.) Benign intramural tumors and cysts of esophagus, 470

N

Neuhof, Harold (with Seley, Gabriel P.) Pulmonary decortication for clotted hemothorax following idiopathic spontaneous hemopneumothorax, 600

O

O'Neal, Lawrence W. (with Ackerman, Lauren V.) Cartilaginous tumors of ribs and sternum, 71
Opaque lobes, observations on division of adhesions in (Coello), 135
Orbison, James A. (with Beattie, Edward J., Jr., Cooke, Francis N., and Paul, John S.) Coarctation of aorta at level of diaphragm treated successfully with preserved human blood vessel graft, 506
Osmotic pressures in tuberculosis: I. Correlation of values with severity of disease, serum protein levels and calculated (Eaton, Sherwood, Kendrick, Eldering, and Daume), 342
Ottosen, Poul (with Beatty, Arch J., and Buckingham, William W.) Resection versus thoracoplasty in treatment of pulmonary tuberculosis, 34
  (with Popp, Carol, Beatty, Arch J., and Buckingham, William W.) One hundred thirty consecutive thoracoplasties—evaluation of results in different types of pulmonary tuberculosis, 202
Oxygen administration: emergency method when intratracheal tube fails with chest open (Brantigan), 532

P

Paraganglioma simulating carcinoma of esophagus (Taylor), 189
Paul, John S. (with Beattie, Edward J., Jr., Cooke, Francis N., and Orbison, James A.) Coarctation of aorta at level of diaphragm treated successfully with preserved human blood vessel graft, 506
Peabody, Homer D., Jr. (with Moersch, Herman J., and Edwards, Jesse E.) Clinically indeterminate pulmonary fibrosis, pathologic study, 519
Pericardium, congenital absence of (Shafiroff), 30
Phreniclasia, angiocardiographic findings in thoracoplasty, artificial pneumoperitoneum, and (McCoy, Steinberg, and Dotter), 149
Pleural sarcoma, surgical treatment of three cases of (Ramstrom and Hellsten), 116
  sclerosis, pathology of—study related to loss of explansivity of lungs and its treatment (Croxatto and Sampetro), 259
Pneumonectomy on respiratory dead space, effect of lung function studies (Fowler and Blakemore), 433
  with replantation of lung in dog for physiologic study (Juvenelle, Citret, Wiles, and Stewart), 111
Pneumonitis of left upper lobe (simulating "middle lobe syndrome" and producing early superior pulmonary sulcus syndrome), nonspecific (Ashe, McDonald, and Clagett), 1
Pneumoperitoneum, and phreniclasia, angiocardiographic findings in thoracoplasty, artificial (McCoy, Steinberg, and Dotter), 149
  in differential diagnosis of diaphragmatic hernia (Clay and Hanlon), 57
Pneumothorax, bronchoscopy in treatment of spontaneous and traumatic (Rubin and Rubin), 377
  in collapse therapy of pulmonary tuberculosis, importance of extrapleural (Cutler), 217
Popp, Carol (with Ottosen, Poul, Beatty, Arch J., and Buckingham, William W.) One hundred thirty consecutive thoracoplasties—evaluation of results in different types of pulmonary tuberculosis, 202
Potts, Willis J. (with Gans, Stephen L.) Anomalous lobe of lung arising from the esophagus, 313
  (with Lewis, James E.) Obstructive emphysema with defect of anterior mediastinum, 438
Potts, W. L. (with Davidson, Horace B.) Bronchiogenic neoplasm masquerading as alveolar cell carcinoma, 402

Presidential address (Blalock), 513
Pulmonary artery in bronchiectasis (Goblel, Gordon, and Digman), 885
  decortication for clotted hemothorax following idiopathic spontaneous hemopneumothorax (Seley and Neuhof), 600
  fibrosis, clinically indeterminate—pathologic study (Peabody, Moersch, and Edwards), 519
  sulcus syndrome, nonspecific pneumonitis of left upper lobe simulating "middle lobe syndrome" and producing early superior (Ashe, McDonald, and Clagett), 1
  tuberculosis, evaluation of results in different types of—one hundred thirty consecutive thoracoplasties (Ottosen, Bopp, Beatty, and Buckingham), 202
    importance of extrapleural pneumothorax in collapse therapy of (Cutler), 217
    resection versus thoracoplasty in treatment of (Beatty, Buckingham, and Ottosen), 34
  veins, anomaly of (Cooke, Evans, Kistin, and Blades), 452

R

Ramstrom, Sven (with Hellsten, Helge) Surgical treatment of three cases of pleural sarcoma, 116
Rasmussen, Richard A. Effective method for bronchial closure, 513
Read, C. Thomas (with Tillett, Wm. S., and Sherry, Sol) Use of streptokinase-streptodornase in treatment of chronic empyema with interpretive discussion of enzymatic actions in field of intrathoracic diseases, 325
  (with Tillett, William S., and Sherry, Sol) Use of streptokinase-streptodornase in treatment of postpneumonic empyema, 275
Resection versus thoracoplasty in treatment of pulmonary tuberculosis (Beatty, Buckingham, and Ottosen), 34
Rib, eosinophilic granuloma of (Kipp and Fisher), 24
Ribs and sternum, cartilaginous tumors of (O'Neal and Ackerman), 71
Rubin, Eli H. (with Rubin, Morris) Bronchoscopy in treatment of spontaneous and traumatic pneumothorax, 377
Rubin, Morris (with Rubin, Eli H.) Bronchoscopy in treatment of spontaneous and traumatic pneumothorax, 377

S

Sampietro, Ruben (with Croxatto, Oscar C.) Pathology of pleural sclerosis: Study related to loss of expansivity of the lungs and its treatment, 259
Sappington, Thomas B., Jr. (with Daniel, Rollin A, Jr.) Accessory diaphragm, 212
Sarcoma, surgical treatment of three cases of (Ramstrom and Hellsten), 116
Scapulectomy, shoulder girdle dysfunction following thoracoplasty combined with partial (Doppelt and Goldberg), 584
Sealy, W. C., Report of two cases of anomalous origin of right subclavian artery from descending aorta, [illegible]
Seley, Gabriel P. (with Neuhof, Harold) Pulmonary decortication for clotted hemothorax following idiopathic spontaneous hemopneumothorax, 600
Serum protein levels and calculated osmotic pressures in tuberculosis: I. Correlation of values with severity of disease (Eaton, Sherwood, Kendrick, Eldering, and Daume), 342
Shafiroff, B. G. P. Congenital absence of pericardium, 30
Sherry, Sol (with Tillett, William S. and Read, C. Thomas) Use of streptokinase-streptodornase in treatment of chronic empyema—with interpretive discussion of enzymatic actions in field of intrathoracic diseases, 325
  (with Tillett, William S., and Read, C. Thomas) Use of streptokinase-streptodornase in treatment of postpneumonic empyema, 275
Sherwood, J. Vincent (with Eaton, Robert M., Kendrick, Pearl L., Eldering, Grace, and Daume, Robert) Serum protein levels and calculated osmotic pressures in tuberculosis: I. Correlation of values with severity of disease, 342
Shoulder girdle dysfunction following thoracoplasty combined with partial scapulectomy (Doppelt and Goldberg), 584
Shumacker, Harris B., Jr. (with Hampton, L. Jennings) Sudden death occuring immediately after operation in patients with cardiac disease, with particular reference to role of aspiration through endotracheal tube and extubation, 48
Sloan, Herbert E. (with Cowley, R. Adams) Posterior mediastinal tracheotomy, 602
  (with Cowley, R. Adams, and Sullenberger, Neil H.) Successful repair of aortic aneurysm with sternal perforation, 159
Steinberg, Israel (with Child, Charles G., III, Harmon, Glen S., and Dotter, Charles T.) Liver herniation simulating intrathoracic tumor, 301
  (with McCoy, Herbert I., and Dotter, Charles T.) Angiocardiographic findings in thoracoplasty, artificial pneumoperitoneum, and phrenic [illegible] 149
Sternal perforation, successful re[illegible] aortic aneurysm with [illegible] and Sullenberger), 159

Stewart, John D. (with Juvenelle, Andre A., Citret, Coleman, and Wiles, Charles E., Jr.) Pneumonectomy with replantation of lung in dog for physiologic study, 111

Streptokinase-streptodornase in treatment of chronic empyema, use of (Tillett, Sherry, and Read), 325
  postpneumonic empyema (Tillett, Sherry, and Read), 275

Stricture formation, heterotopic gastric mucosa in esophagus with ulceration and (Bosher and Taylor), 306

Sullenberger, Neil H. (with Cowley, R. Adams, and Sloan, Herbert E.) Successful repair of aortic aneurysm with sternal perforation, 159

T

Takaro, Timothy (with Clagett, O. Theron) Cavernous hemangioma of thoracic wall associated with clinical evidence of arteriovenous fistulas, 444

Tantalum mesh gauze, reconstruction of defects of thoracic wall with (Ada and Hevenor), 125

Taylor, Frederick H. Paraganglioma simulating carcinoma of esophagus, 189
  (with Bosher, Lewis M., Jr.) Heterotopic gastric mucosa in esophagus with ulceration and stricture formation, 306

Thoracic gunshot wound, bullet embolus to left femoral artery following (Keeley), 608
  wall associated with clinical evidence of arteriovenous fistulas, cavernous hemangioma of (Takaro and Clagett), 444
  with tantalum mesh gauze, reconstruction of defects (Ada and Hevenor), 125

Thoracoplasties, one hundred thirty consecutive—evaluation of results in different types of pulmonary tuberculosis (Ottosen, Bopp, Beatty, and Buckingham), 202

Thoracoplasty, artificial pneumoperitoneum, and phreniclasia, angiocardiographic findings in (McCoy, Steinberg, and Dotter), 149
  combined with partial scapulectomy, shoulder girdle dysfunction following (Doppelt and Goldberg), 584
  in treatment of pulmonary tuberculosis, resection versus (Beatty, Buckingham, and Ottosen), 34

Thorax, tracheotomy, useful procedure in thoracic surgery, with particular reference to its employment in crushing injuries of (Carter and Giuseffi), 495

Tillett, William S. (with Sherry, Sol, and Read, C. Thomas) Use of streptokinase-streptodornase in treatment of postpneumonic empyema, 275
  (with Sherry, Sol, and Read, C. Thomas) Use of streptokinase-streptodornase in treatment of chronic empyema—with interpretive discussion of enzymatic actions in field of intrathoracic diseases, 325

Trachea, mobilization of arch of aorta in order to reach bifurcation of (Monod, Babou, Kateb, and Ghazi), 492

Tracheal and bronchial defects with stainless steel, experimental reconstruction of (Bucher and Burnett), 572
  segments preserved by lyophilization, homotransplantation of—experimental study (Marrangoni), 398

Tracheotomy, posterior mediastinal (Sloan and Cowley), 602
  useful procedure in thoracic surgery, with particular reference to its employment in crushing injuries of thorax (Carter and Giuseffi), 495

Tuberculosis, blood volume and extracellular fluid in (Hay and Jenkins), 42
  serum protein levels and calculated osmotic pressure in: I. Correlation of values with severity of disease (Eaton, Sherwood, Kendrick, Eldering, and Daume), 342

Tumor, liver herniation simulating intrathoracic (Child, Harmon, Dotter, and Steinberg), 391

Tumors and cysts of esophagus, benign intramural (Myers and Bradshaw) 470
  of lung, primary solitary neurogen
  (Diveley and Daniel), 194

V

Ventricular pressures in animals, effect
decreasing amount of lung
right (Carlson, Charbon, Cha
and Adams), 621

W

Wegelius, C. (with Keyes, T. F., and Lind, J.
Diagnostic value of dynamic studi
in angiocardiography, evaluation
new rapid technique, 164

Wiles, Charles E., Jr. (with Juvenelle, Andr
A., Citret, Coleman, and Stewar
John D.) Pneumonectomy with r
plantation of lung in dog for physic
logic study, 111